Right ventricular infarction : its detection by electrocardiography and its effect on right ventricular ejection fraction

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Maastricht, 1984
Druk: Leiter-Nypels bv

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Proefschrift

Ter verkrijging van de graad van doctor in de Geneeskunde aan de Rijksuniversiteit Limburg te Maastricht, op gezag van de Rector Magnificus Prof.Dr. H.C. Hemker, volgens besluit van het College van Dekanen in het openbaar te verdedigen in de aula van de universiteit op 6 september 1984 des namiddags te vier uur.

door

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Het verschijnen van dit proefschrift werd mogelijk gemaakt door steun van de Nederlandse Hartstichting en Rescar (stichting ter bevordering research cardiologie) Maastricht.

Aan Anneke
Jeroen en Manon
en mijn ouders

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Chapter 1

Introduction

Right ventricular infarction was initially a diagnosis only made by pathological observation. In a review of 2,000 consecutive autopsies Wartman and Hellerstein (1) in 1948 described 22 instances of right ventricular infarction out of 164 cases of myocardial infarction (13,8% prevalence). In 18 hearts left and right ventricular infarction was observed while 4 hearts had isolated right ventricular infarction. Wade (2) examined 19 postmortem hearts with right ventricular infarction and found occlusion of the right coronary artery in 18 specimens. Isner and Roberts (3) reported an incidence of 14% of right ventricular infarction in a group of 236 patients with a transmural infarction at necropsy. Right ventricular infarction was only found when transmural infarcts of the posterior septum was present. The incidence of right ventricular dilatation in combined inferior wall and right ventricular infarction was three times that found in the absence of right ventricular involvement ($p < 0.05$). The characteristic hemodynamic manifestation of right ventricular infarction is a systemic venous hypertension in the presence of low left sided filling pressures (4). It is important to recognize this clinical entity because it requires specific therapy in the form of aggressive volume administration.

Although invasive hemodynamic monitoring of patients with acute myocardial infarction has gained popularity in recent years, the technique is not available in every hospital. Non-invasive techniques to make the diagnosis of right ventricular infarction are therefore required. Better knowledge of the syndrome has been gained by the use of echocardiography (5, 6) and cardiac scintigraphy (5, 6, 7, 8).

As electrocardiography is inexpensive and readily available, the purpose of this study was to assess the value of additional right precordial leads for diagnosing right ventricular infarction and to assess its value in locating the coronary artery lesion which is responsible for inferior myocardial infarction or recognize a critical stenosis in the right coronary artery. The predictive value of diagnosing right ventricular infarction with regard to subsequent AV-block was also studied. Lastly, the effect of right ventricular infarction on the ejection fraction of the right ventricle in the acute phase of myocardial infarction was evaluated.

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Chapter 2

Methode

This chapter describes the methods used in all the reported studies. Emphasis will be placed upon the right chest wall electrocardiogram, the radionuclide techniques applied and the invasive investigations used. Also the value and limitations of these techniques will be discussed.

a. Right chest wall electrogram

A standard 12-lead electrocardiogram is recorded from all patients admitted to our department using either a Siemens Elema Mingograph 62 6 channel recorder or a Marquette 4000 3 channel recorder with standard American Heart Assocation (AHA) filter settings (1).
Leads I, II, III, aVR, aVL, aVF and the unipolar precordial leads V_1-V_6 are recorded together with right sided chest leads.
Standard positions are used for lead V_1-V_6 and mirror image position for the right chest leads V_1R-V_6R (table 1). In this way, V_1 and V_2 are equal to the right sided leads V_2R and V_1R respectively. The placement of all the chest leads is illustrated in figure 1.

Table 1

Lead No.	Anatomical position
V_1	4th ICS at right sternal border
V_2	4th ICS at left sternal border
V_3	Midway between V_2 and V_4
V_4	5th ICS in left midclavicular line
V_5	At level of V_4 in left anterior axillary line
V_6	At level of V_4 in left midaxillary line
V_1R	As V_2
V_2R	As V_1
V_3R	Midway between V_2R and V_4R
V_4R	5th ICS in right midclavicular line
V_5R	At level of V_4R in right anterior axillary line
V_6R	At level of V_4R in right midaxillary line

Abbreviations: ICS = Intercostal space

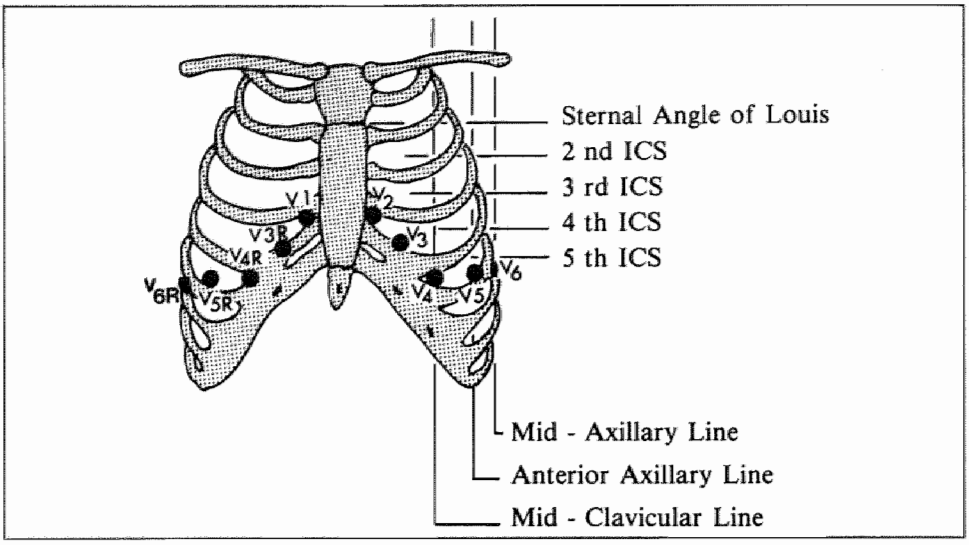

Figure 1 The placement of the right and left precordial leads.

The normal pattern of the right chest wall leads:

Before studying the pattern of electrocardiographic changes in the right chest wall leads in patients with myocardial infarction we defined the normal pattern in these leads. A standard 12-lead electrocardiogram and 6 right chest wall leads were recorded in 30 volunteers. None of the 30 volunteers (18 male, age range 22 to 35 years) had a history of ischemic heart disease. All had a normal 12-lead standard electrocardiogram. The pattern in the right chest wall leads was as follows: In lead V_1R (V_2) an ST-segment elevation $\geqq$ 1 mm was seen in 20 volunteers (18 males and 2 females). They all showed the pattern of early ventricular repolarization with ST-segment elevation at the J-point. The high incidence of early repolarization is probably accounted for by the low mean age (26 years) of this group.
In lead V_2R (V_1) ST-segment elevation $\geqq$ 1 mm was observed in two subjects. None of the volunteers showed ST elevation $\geqq$ 1 mm in lead V_3R, V_4R, V_5R and V_6R. A QS-pattern was seen in V_3R and V_4R in 2 volunteers. In 27 a negative T-wave was observed in the right precordial leads. We conclude that in normals the right chest wall leads do not show ST elevation $\geqq$ 1 mm in V_2R, V_3R, V_4R, V_5R and V_6R. A QS-pattern in lead V_3R and V_4R and ST-segment elevation in V_2R (V_1) is seen infrequently. The majority of normals show negative T-waves in the right precordial leads. Our volunteers represent an age group younger than our patients with myocardial infarction (mean age 57 years). We were unable to find an age-matched control group of patients free from cardiac symptoms.

b. Radionuclide techniques used in this study

1. Pyrophosphate imaging for infarct detection

In 1974 Bonte et al (2) reported that technetium 99m labeled to stannous pyrophosphate localized in acutely infarcted myocardium in animal studies. This technique has also proved sensitive for identifying acutely infarcted myocardial tissue in humans (3, 4). The mechanism of Tc99m stannous pyrophosphate binding in such tissues has yet to be clearly defined.

Technique

Technetium 99m stannous pyrophosphate was the first agent used to image acute myocardial infarction in both the animal and the human heart. Dosages of 15 mC to 20 mC technetium 99m tagged to 5 mg of stannous pyrophosphate were most commonly used for acute myocardial necrosis imaging in our studies. Imaging of the heart was started at between 60 and 90 minutes following the injection of the radionuclide. The postinfarction timing of the study was 36-72 hours after the onset of chest pain.

Good labeling of the phosphate compounds with technetium 99m is required to prevent poor clearance of the tracer from the blood pool. This could simulate diffuse tracer concentration in the myocardium and give rise to positive interpretation. Tc99m stannous pyrophosphate must be injected within 1 hour of its preparation to minimize release of the Tc99m from its binding compound.

Equipment

This radionuclide technique requires a gamma camera and a computer to process the images. The gamma camera should have a high resolution, high sensitivity or an all purpose collimator. Field uniformity is essential as warm areas in the field can be erroneously interpreted as a positive myocardial scintigram.

In our studies we used either a Philips camera with a parallel hole all purpose collimator interfaced to a Philips mini- computer P855M or an Ohio Nuclear Sigma 420 mobile gamma camera interfaced to a MCS 560 mobile Ohio Nuclear computer. We used a 20% window, which was set symmetrically around the 140 KEV photopeak of technetium 99m.

Positioning

Patients were routinely imaged in the supine position using the anterior and 45° left anterior oblique projections. Left lateral views were obtained with the patient lying on his right side, to minimize the distance between the heart and the camera and thus preserve a high resolution. More than 600,000 counts were collected in each view, 60 to 90 minutes after the injection of technetium 99m stannous phosphate i.v.

Image interpretation

The sensitivity and specificity of technetium 99m stannous phosphate scintigraphy is dependent on camera field uniformity, radiopharmaceutical stability, tagging efficiency and postinjection and postinfarction timing.

Scintigrams are commonly graded 0 to 4+ depending on the activity over the myocardium. Grading refers to the intensity of the suspected lesion and not to its size. O represents no activity and 1+ indicates minimal activity believed to be in blood pool or chest wall. 2+ indicates definite myocardial activity, 3+ activity equal to bone activity and 4+ activity greater than bone activity. This system has been shown to have a good correlation with electrocardiographic and enzymatic criteria for determining the presence of infarction. Inferior myocardial infarcts are visualized as platelike areas of activity extending to the left of the sternum in anterior views.

Parkey (5) found this technique to be highly accurate. Only 4% of false negative scintigrams were found while false positive results range from 8 to 12% when the ECG is used as the gold standard. The false positive scintigrams were found in patients with unstable angina.

Anatomic location of the right and left ventricle

To determine the borders of the right and left ventricle a bolus 5 mCi technetium was injected with the patients in the left anterior oblique position (45°). A dynamic flow study was recorded using a frame rate of one per second. The right atrium, right ventricle, lungs, left atrium, left ventricle and aorta were visualized separately. This enabled a region of interest (ROI) representing the right ventricle to be determined. This ROI was then superimposed on the 45°left anterior oblique view of the technetium 99m stannous phosphate image to asses the presence of activity localized to the right ventricle free wall.

Limitations

Areas of acute myocardial infarction cannot be reliably defined by scintigraphy with intravenous injection of technetium 99m stannous pyrophosphate for at least 24 hours after the onset of chest pain. Earlier imaging with this radionuclide can only be achieved if direct intracoronary injection is performed.

Using the ECG combined with serial enzyme estimation as the "gold standard" for infarct detection, technetium 99m stannous pyrophosphate imaging is found to result in 4% false negative scintigrams. In our studies there were no false negatives, probably because only patients with transmural infarction and an enzyme rise to at least twice normal values were included. Though infarct imaging requires only small and therefore inexpensive, quantities of technetium 99m (15-25 mCi) high quality data cannot be obtained without a reliable gamma camera and computer system. This equipment is relatively expensive and limits the availability of this technique.

d. Multigated cardiac blood pool imaging

In this technique, images of the blood pool in the heart at fixed points in the cardiac cycle are obtained by synchronizing the recording of scintillation data to an indicator of cardiac contraction, such as the electrocardiogram. Using a physiological signal that occurs at a fixed time in relation to the mechanical activity of the heart to gate the recording of scintillation events, repetitive sampling during specific phases of many consecutive cardiac cycles enables images of reasonable count density to be recorded.

A high-quality blood pool label is critical for the acquisition of high resolution data in a short imaging interval. The relationship between activity in the cardiac blood pool and that in the pulmonary blood pool background will determine the accuracy with which the borders of the chambers can be defined for a given count density of data. Technetium 99m currently appears to be the most appropriate radionuclide for use with the scintillation camera. The binding agents that have been suggested as blood pool labels include both albumen (6) and red blood cells (7). Albumen has a relatively large distribution volume compared to that of red blood cells. This volume includes the liver and to a lesser extent the lungs. Images recorded with this radiopharmaceutical will therefore have a lower heart to lung activity ratio than those recorded with labeled red blood cells (8). However, even with the technetium 99m labeled red blood cells a heart to lung ratio of only 3:1 can be achieved.

Both in vivo and in vitro methods have been proposed to achieve cell labeling. There is no evidence of any difference between there methods in terms of the results obtained in the clinical situation (7).

In our clinic we use the method described by Pavel et al (9). Fifteen to 30 minutes after an intravenous injection of a stannous pyrophosphate solution (5 mg) a second intravenous injection with 15-20 mCi technetium 99m pertechnetate is given. The technetium 99m binds almost instantaneously to the globin portion of the hemoglobin with a labeling efficiency of well over 90% and does not clear from the cells for several hours. As described by Alderson (10) the unlabeled portion of the injected does appears to clear rapidly from the body in the urine; thus a high target to background activity ratio is maintained over the heart.

Imaging technique

We used a Philips camera with a parallel hole-all-purpose collimator interfaced to a Philips minicomputer (P855M). Data were collected in three different views: anterior, left lateral and left anterior oblique (30-55°). In the latter view, the best separation between the right and left ventricle could be achieved. We used a 20% window which was set symmetrically around the 140 KEV photopeak of technetium. Data were collected and stored on disc in synchronized frame mode by dividing an average cardiac cycle into 32 frames with 64 x 64 pixel elements.

Processing methods used in our studies

Arrhythmia filtering program

Cycles that were either too long or too short with respect to the mean were rejected. The limits of acceptable cycle length were defined as + 20% of the average RR-interval. Because of the variation in heart rate, the acquisition time for each frame was not constant. A correction factor was therefore derived from the RR-interval of each cycle. Acquisition time for each view is 300 seconds.

Processing methods for LVEF and RVEF calculation

After acquisition of the data a semi-automatic program was initiated to perform a three dimensional filter process in space (X, Y) and time (T). This filter system enables the reservation of important information regarding organ structure and movement. After completion of the filter program, a region of interest was placed around the left or right ventricle in the end-diastolic frame. Edge detection of the ventricles was performed by means of the second derivative of count activity.
The second derivative was determined with a 7 point Laplace operator in 4 independent directions. This algorithm can be used to construct a closed contour around either the right or left ventricle. A variable region of interest was thus constructed for each of the 32 frames representing a cardiac cycle. Background correction was determined from the end-systolic frame. RVEF and LVEF were calculated according to

$$EF = \frac{EDC - ESC \times 100\%}{EDC}$$

EDC = end-diastolic counts,
ESC = end-sysolic counts

To determine normal limits for LVEF and RVEF 20 patients were studied. Ten were male and the age range was 28-63 years. Ten of these patients had no cardiac history, and were studied just before the first treatment with adriamycin for breast cancer. The remaining patients were studied 2 days after diagnostic heart catheterisation for the investigation of chest pain. None had a history of myocardial infarction and in all ten the ventriculograms were judged to be normal. The right and left ventricular ejection fraction were determined in these patients by multigated blood pool imaging. The lower limits of normal LVEF and RVEF were then defined as the mean minus twice the standard deviation of the ejection fraction in the group. Values of 60% for LVEF and 40% for RVEF were found.

Validation of the ejection fraction calculation

The ejection fraction calculated from the left ventriculogram is still regarded as the "gold standard". This method to determine ejection fraction will be described in the section on angiographic techniques. To validate the results of ejection fraction by nuclear imaging, we assessed the correlation between values obtained with this technique and those calculated from the left ventriculogram in the same group of

patients. Forty consecutive patients (32 male, age range 34-68 years) who were admitted for cardiac catheterisation were also studied by nuclear angiography. There was close correlation between the values of LVEF determined by the two methods (r = 0.84) (fig. 2).

In the absence of any other reliable means of determining RVEF, it is impossible to validate the values obtained by nuclear techniques. The calculation of the right ventricular ejection fraction in a multigated study is complicated by the changing overlap of the right atrium. Because there are no reasons to believe that there is a difference between the overlap of the right atrium in patients with or without right ventricular infarction, we believe that we were justified in comparing the value of RVEF, derived by multigated nuclear angiography in the two groups (chapter 5). Because we used a "semi"-automatic program for calculation of the right and left ventricular ejection fraction the inter and intraobserver variability is zero.

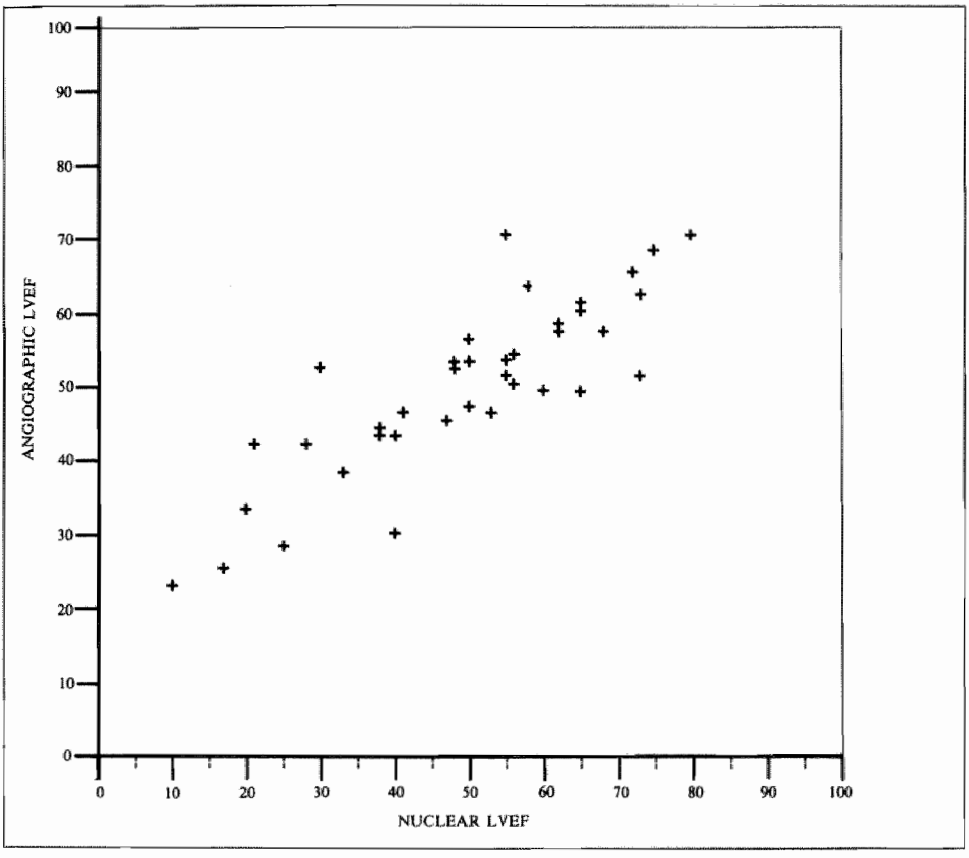

Figure 2 The relation between the radionuclide and angiographic left ventricular ejection fraction.

e. Angiographic techniques used in our studies

After local anaesthesia with lidocain 2%, a French 8 Cordis pigtail catheter was introduced percutaneously into a femoral artery using the Seldinger technique. Following hemodynamic measurements, left ventriculography was performed using 60° LAO (left anterior oblique) and 30° RAO (right anterior oblique) projections. In our catheterisation laboratory we have a Siemens U-arm angioscope. Cine films were taken at 50 frames per second by a 35 mm Arriflex camera mounted on a 9 inch image intensifier. The films were developed using an Oude Delft processor.

Measurement of the ejection fraction on the left ventriculogram

The films were projected on an Arriflex RGT screen. A transparant sheet of paper was fixed on the screen and the end-systolic and end-diastolic contours in the right anterior oblique position drawn on this paper. The left ventricle ejection fraction was calculated from these contours by means of Hewlett Packard digitizer (9874A) and computer (9830A).

Coronary arteriography

Following left ventriculography, coronary angiography was performed using Judkins catheters. The right coronary artery was studied in 2 projections: LAO 60° and RAO 30°. The left coronary artery was studied in 4 projections: LAO 60°, LAO with craniocaudal tilt, RAO 30° and RAO with caudocranial tilt. The resulting cine films were projected using a Tagarno 35CX. A visual assessment of the degree of stenosis was determined by 2 observers. Callipers measurement was used to resolve any interobserver disagreement.

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Chapter 3

Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction

by

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Published in the British Heart Journal 1983, 49: 368-72 and in part presented at the 54th Annual Scientific Meeting of the American Heart Association, Dallas, November 1981

Summary

To study the value of the electrocardiogram in diagnosing right ventricular involvement in acute inferior wall myocardial infarction the electrocardiographic findings were analysed in 67 patients who had had scintigraphy to pin-point the infarct. All 67 patients were consecutively admitted because of an acute inferior wall infarction. A 12 lead electrocardiogram with four additional right precordial leads (V_3R, V_4R, V_5R and V_6R) was routinely recorded on admission and every 8 hours thereafter for three consecutive days. Thirty-six to 72 hours after the onset of chest pain a 99m-technetium pyrophosphate scintigraphy and a dynamic flow study were performed to detect right ventricular involvement, which was found in 29 of the 67 patients (43%). ST-segment elevation $\geqq$ 1 mm in leads V_3R, V_4R, V_5R and V_6R is a reliable sign of right ventricular involvement. ST-segment elevation $\geqq$ 1 mm in lead V_4R was found to have the greatest sensitivity (93%) and predictive accuracy (93%). The diagnostic value of a QS pattern in lead V_3R and V_4R or ST elevation $\geqq$ 1 mm in lead V_1 was much lower. ST-segment elevation in the right precordial leads was short lived, having disappeared within 10 hours after the onset of chest pain in half of our patients with right ventricular involvement. When electrocardiograms are recorded in patients with an acute inferior wall infarction within 10 hours after the onset of chest pain, additional right ventricular infarction can easily be diagnosed by recording lead V_4R.

Until recently, the diagnosis of right ventricular infarction was only possible at necropsy. In 1948, in their review of 2000 consecutive necropsies, Wartman and Hellerstein (1) described 22 instances of right ventricular infarction out of 164 cases of myocardial infarction. At necropsy of 19 hearts with right ventricular infarction, Wade (2) found that the major damage was located on the posterior wall of the heart. In all 19 patients the electrocardiogram had shown an inferior wall myocardial infarction.

In 1974 Cohn et al (3) reported characteristic haemodynamic changes in six patients with an inferior wall myocardial infarction, who also had right ventricular infarction. Sharpe et al (4) observed that six out of 15 patients with an inferior wall infarction showed abnormal technetium pyrophosphate uptake in the right ventricle. In a study of patients with an acute inferior wall myocardial infarction Wackers et al (5) found that in 37% the technetium pyrophosphate uptake also showed involvement of the right ventricle.

Erhardt et al (6) in 1976 described the value of a right precordial lead V_4R in diagnosing right ventricular involvement. They compared their data with necropsy findings and found right ventricular infarction in 9 out of 18 patients (50%). These data have subsequently been confirmed by other investigators (7). Recently, Chou et al (8) described the value of lead V_1 in diagnosing right ventricular infarction in 11 pa-

tients. The diagnosis of right ventricular infarction was proven at necropsy and supported by hemodynamic findings. Morgera et al (9) in a preliminary report described the value of a QS pattern in lead V_3R and V_4R in diagnosing right ventricular infarction.
The incidence of abnormal hemodynamic findings suggesting right ventricular infarction is much lower than findings pointing to right ventricular involvement at necropsy or scintigraphy. Therefore we decided to compare the value of the different electrocardiographic criteria in diagnosing right ventricular infarction in patients with inferior wall infarction when additional right ventricular involvement was shown by 99mTechnetium pyrophosphate scintigraphy.

Patients and methods

Studies were made on 67 consecutive patients (56 men, 11 women), admitted because of an acute inferior wall infarction. Four had a documented myocardial infarction in the past, in two on the anterior and in two on the inferior wall.
Patients were admitted half an hour to 30 (mean five) hours after the onset of chest pain. Ages ranged from 39 to 80 (mean 57 $\pm$ 9.4) years. The diagnosis of acute inferior wall infarction was based on the clinical history, a characteristic enzyme pattern of CPK and AST values, and the appearance of new pathological Q waves in the inferior leads (II, III and AVF). On admission and every eight hours during the next three days a 12 lead ECG and four additional right precordial leads were recorded (Fig. 1).

The amount of ST elevation in lead V_1, and in leads V_3R, V_4R, V_5R and V_6R (these leads are the mirror image of leads V_3, V_4, V_5 and V_6), was measured and the duration of its presence noted. We also looked for the presence of a QS pattern in leads V_3R and V_4R.
At the time of the electrocardiographic registrations blood was taken to determine the values for CPK and AST. Normal values for CPK and AST in our laboratory are, respectively, less than 240 and 40 U/l. None of the 67 patients had clinical signs of right ventricular infarction on physical examination. No hemodynamic monitoring was performed in any of the patients. Thirty-six to 72 hours after the onset of chest pain a 99mTechnetium pyrophosphate scintigraphy was performed. A Philips scintillation camera with a general all purpose parallel hole collimator interfaced to a PDS computer system or a Ohio Nuclear Sigma 420 mobile gamma camera with a general all purpose parallel hole collimator interfaced to a MCS 560 mobile computer system was used for all studies. One hour after the injection of 15 to 20mCi of 99mTechnetium pyrophosphate, the anterior, the left lateral, and the 45° left anterior oblique views were recorded with a general all purpose parallel hole collimator

(Fig. 2). Each view contained at least 600,000 counts. After the last view was recorded, which was always the 45° left anterior oblique, a small bolus of 99mTechnetium was injected without moving the patient or the collimator.
Simultaneously a dynamic flow study was performed, using frames of one second to visualise separately the right and left ventricle (Fig. 3).
A region of interest was placed around the right and left ventricle and these regions of interest were superimposed on the left anterior oblique view to verify whether there was right ventricular involvement (Fig. 4).
The radionuclide data were analysed by two independent observers without knowledge of the clinical data. The 99mTechnetium pyrophosphate scintigraphy was judged to be positive when there was myocardial uptake. Right ventricular involvement was considered to be present if definite myocardial uptake was seen in the region of interest of the right ventricle.

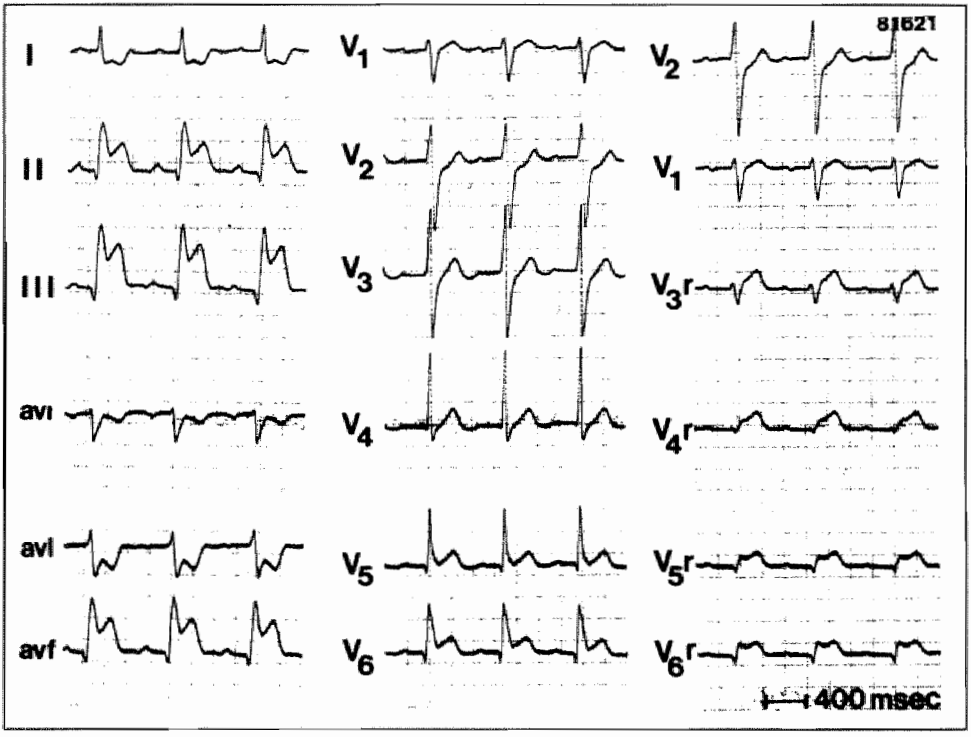

Figure 1 Leads I, II, III, AVR, AVL, AVF, leads V_1, V_2, V_3, V_4, V_5, V_6 and leads V_2, V_1, V_3R, V_4R, V_5R and V_6R are recorded simultaneously. This electrocardiogram shows an acute inferolateral wall myocardial infarction. The right precordial leads show ST-segment elevation in leads V_3R, V_4R, V_5R and V_6R, in the absence of a QS-pattern in lead V_3R or V_4R or ST-segment elevation in lead V_1.

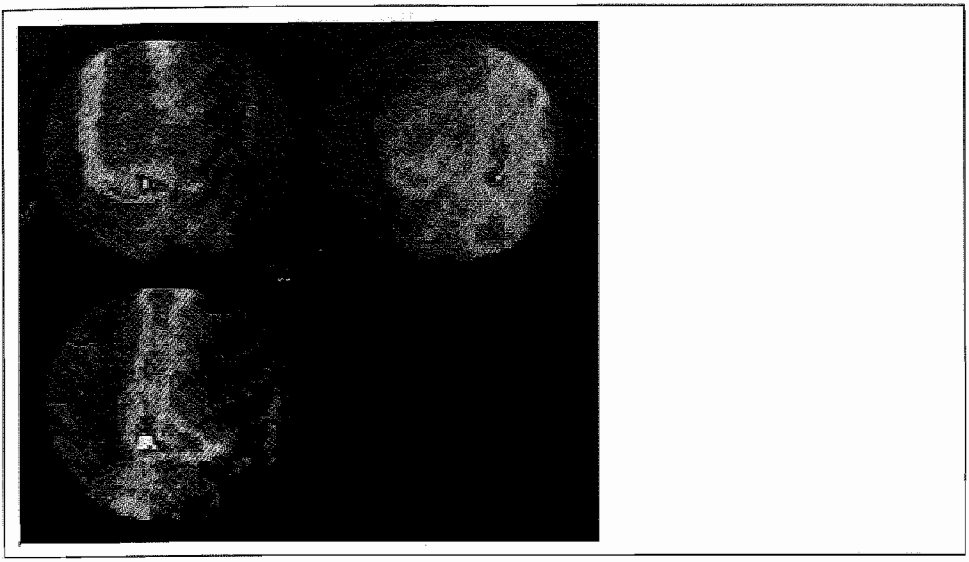

Figure 2 An example of a 99mTechnetium pyrophosphate scan in three different views. Top left the 45° left anterior oblique view. On the right, the left lateral, and below the anterior view. Apart from uptake in the sternum, spine, and ribs, pathological 99mTechnetium pyrophosphate uptake is seen in the inferior wall of the myocardium.

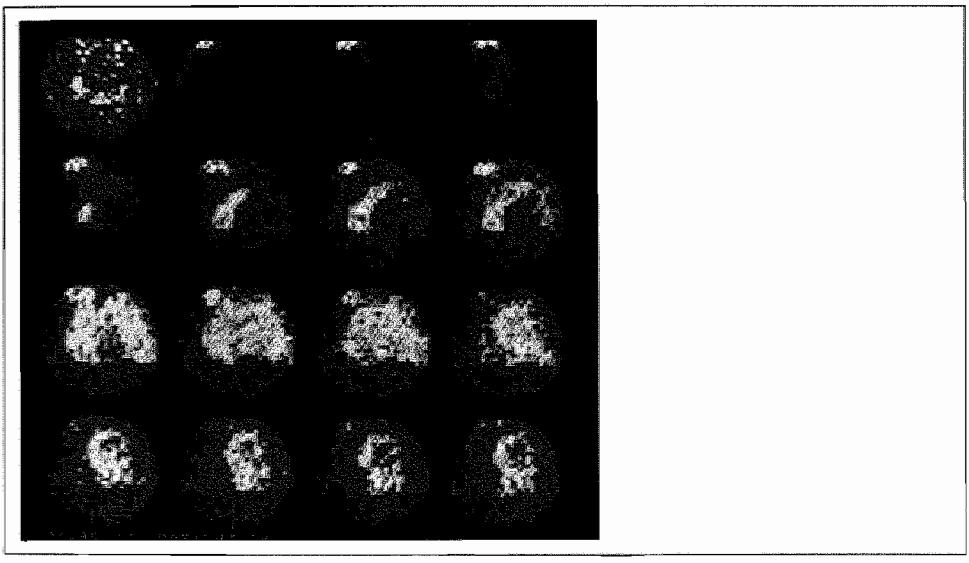

Figure 3 An example of a dynamic flow study. The activity can be followed from the superior caval vein to the right ventricle, the lungs, the left ventricle, and the aorta.

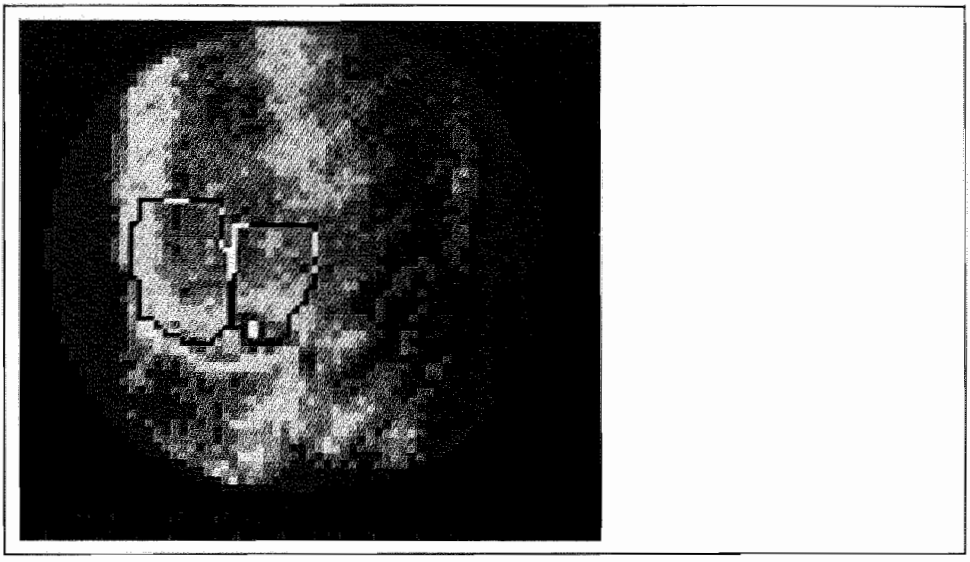

Figure 4 The 45° left anterior oblique view on which is superimposed the region of interest drawn around the right and left ventricle. Definite involvement of the right ventricle is seen.

Results

All 67 patients had pathologic uptake of 99mTechnetium pyrophosphate in the inferior wall. Twenty-nine patients (43%) also had right ventricular involvement. Twenty-one patients had ST-segment elevation $\geqq$ 1 mm in lead V_3R. Only in one patient did the scan not show right ventricular involvement. Twenty-nine patients had ST-segment elevation in lead $V_4R \geqq 1$ mm. In two of this group there was no right ventricular involvement on the 99mTechnetium scan. In only 3 of the 29 patients with ST-segment elevation in lead $V_4R \geqq 1$ mm did this elevation persist for more than 72 hours.

In 14 patients the duration of significant ST elevation lasted less than 10 hours after onset of chest pain. In the two patients without ST elevation in lead V_4R but with a positive 99mTc scan, 15 to 30 hours, respectively, had elapsed before arrival in hospital.

In addition, in 29 patients there was ST-segment elevation in lead V_5R. Three of them had no pathological 99mTechnetium uptake in the right ventricle. Twenty-seven patients had ST elevation $\geqq$ 1 mm in lead V_6R. As shown in table 1, in 3 cases there was disagreement between the electrocardiographic and the scintigraphic data.

Sixteen patients with a QS-pattern in leads V_3R and V_4R had right ventricular invol-

vement on the scan. Of the 29 patients with scintigraphic right ventricular infarction only 8 had ST elevation in lead $V_1 \geqq 1$ mm. A false positive QS-pattern in lead V_3R and V_4R, or ST elevation in lead V1 was seen in 5 and 3 patients, respectively. Table 2 shows the sensitivity, specificity and predictive accuracy of ST elevation $\geqq$ 1 mm in leads V_1, V_3R, V_4R, V_5R, V_6R and of a QS-pattern in leads V_3R and V_4R.

Table 1

Presence of ST segment elevation in leads V_3R, V_4R, V_5R and V_6R in patients with acute inferior wall infarction with and without right ventricular involvement.

ST elevation > 1 mm	**V_3R**	**V_4R**	**V_5R**	**V_6R**
Positive pathological 99mTc uptake in right ventricle	20	27	26	24
Negative pathological 99mTc uptake in right ventricle	1	2	3	3
No ST elevation $\geqq$ 1 mm				
True negative	37	36	35	35
False negative	9	2	3	5

Table 2

Sensitivity, specificity and predictive accuracy of ST-segment elevation $\geqq$ 1 mm in leads V_1, V_3R, V_4R, V_5R and V_6R and a QS-complex in leads V_3R or V_4R in diagnosing right ventricular involvement in patients admitted because of an acute inferior myocardial infarction.

ST-segment elevation $\geqq$ 1 mm	**Sensitivity**	**Specificity**	**Predictive accuracy**
V_1	28	92	73
V_3R	69	97	95
V_4R	93	95	93
V_5R	90	92	90
V_6R	83	92	89
QS-pattern			
V_3R	55	87	76
V_4R	55	87	76

The mean peak AST value of all the 67 patients was 255 ± 120 U/l. In the 29 patients with right ventricular infarction the mean AST was 271 ± 120 U/l and in the 38 patients without right ventricular infarction 243 ± 118 U/l. This difference has no statistical significance. The mean peak CK value of all the patients was 2262 U/l and here also there was no statistical significant difference between the patients with and without right ventricular infarction (respectively 2321 ± 1548 U/l and 2177 ± 1418 U/l). Only one of the patients died in hospital, suddenly on the third day after admission, and cardiac tamponade was found at autopsy.
This patient had a positive scintigraphy for right ventricular involvement and ST elevation > 1 mm in lead V_3R, V_4R, V_5R and V_6R, but no QS-pattern in lead V_3R or V_4R, nor ST elevation in V_1. At necropsy apart from an inferoposteroseptal infarction right ventricular involvement was also found.

Discussion

Since Cohn et al (3) in 1974 reported on the value of recognizing additional right ventricular infarction in patients with inferior wall infarction, interest in diagnosing this abnormality has grown. Since then it has become clear, however, that the clinical and hemodynamic features of right ventricular infarction are found in only a small percentage of patients showing right ventricular infarction at necropsy or during cardiac scintigraphy (10). In our series 43% of patients with an inferior wall myocardial infarction had right ventricular involvement. Maximal enzyme values and clinical course during admission were not different in patients with and without additional right ventricular infarction. Because of the high incidence of atrioventricular nodal conduction disturbances in patients with inferior wall myocardial infarction with associated right ventricular infarction (11), we feel that it is desirable to have an easy and cheap tool to detect right ventricular involvement. Our results indicate that lead V_4R is the single most valuable electrocardiographic lead to detect right ventricular involvement. It is of importance to stress, however, that the duration of ST-segment elevation is short, disappearing in less than 10 hours in 48% of our patients with right ventricular infarction.
This indicates the necessity for early recording of the right precordial leads. Our data indicate that ST elevation in lead V_1 or a QS-pattern in V_3R and V_4R does not have the same diagnostic value as ST-segment elevation in leads V_3R, V_4R, V_5R and V_6R. In addition, a combination of V_4R and V_1 or V_4R and a QS-pattern in V_3R or V_4R does not improve the diagnostic accuracy of the electrocardiogram in making the diagnosis of right ventricular infarction.
The most consistent finding in our patients with right ventricular infarction was the ST-segment elevation in the right precordial leads. Necropsy data have shown that right ventricular infarction is seen almost exclusively in patients with inferior wall

infarction in combination with posteroseptal involvement. Perhaps the ST-segment elevation in the right precordial leads is an expression of posteroseptal involvement. The finding of a QS-pattern in V_4R and V_3R requires further study. Again this could be the result of the loss of septal forces from the infarcted posteroseptal area. The incidence of a QS-pattern in right precordial leads in patients without myocardial infarction is, however, not known. From our data and those of others (6,7, 12) we conclude that the single recording of lead V_4R in order to see an ST-segment elevation of equal to or more than 1 mm is the most reliable way to diagnose right ventricular involvement in patients admitted because of an acute inferior myocardial infarction.

Acknowledgement

We would like to thank Dr. Frans J. Wackers, Department of Internal and Nuclear Medicine, University Hospital, Burlington, Vermont, for his support and advice during the preparation of this manuscript.

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Chapter 4

Value or lead V_4R for recognition of the infarct coronary artery in acute inferior myocardial infarction

by

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Published in the American Journal of Cardiology 1984, 53: 1538-1541.

Summary

In 84 patients with an acute inferior wall myocardial infarction (MI) admitted within 10 hours after the onset of chest pain a right precordial lead V_4R was recorded in addition to the standard 12-lead ECG. The presence or absence of ST-segment elevation in lead V_4R during the acute phase of MI was correlated with results of coronary angiography, 2-26 (mean 10) weeks after the acute infarction. Patients were divided into those with a critical stenosis or occlusion proximal (27 patients) or distal (36 patients) to the right ventricular branch of the right coronary artery (RCA) or in the circumflex coronary artery (CA) (21 patients). The presence of ST-segment elevation $\geqq$ 1 mm in lead V_4R has a sensitivity of 100% and a specificity of 87% for occlusion of the RCA above the level of the first branch to the right ventricle, while its predictive accuracy is 92%. In contrast, 7 out of 36 patients with a distal occlusion of the RCA showed ST-segment elevation $\geqq$ 1 mm in lead V_4R ($p < 0.001$). None of 21 patients with occlusion of the CA had ST-elevation in V_4R. The absence of ST-segment elevation $\geqq$ 1 mm in lead V_4R appears to exclude proximal occlusion of the RCA. Therefore, recording of right precordial lead V_4R within 10 hours after onset of acute inferior wall MI provides a reliable and simple means to predict the location and level of occlusion in the infarct vessel. These findings are of clinical relevance when emergency procedures (intracoronary streptokinase infusion, balloon dilatation, emergency surgery) are considered.

Since Cohn et al (1) described the syndrome of predominant right ventricular dysfunction in the setting of an acute inferior wall myocardial infarction (MI) the interest of recognizing right ventricular infarction (RVI) with non-invasive techniques has grown. The reason being the therapeutic implications of separating patients in those with right ventricular dysfunction and those with the more usual clinical presentation of left ventricular dysfunction. Electrocardiographic diagnosis of RVI using right precordial leads has been correlated with autopsy, scintigraphic, echo-cardiographic and hemodynamic data (2-7). ST-segment elevation $\geqq$ 1 mm in right precordial lead V_4R (2-6) was found as the best electrocardiographic predictor of RVI in the setting of an acute inferior MI. RVI in the setting of an acute inferior wall MI is believed to occur when the RCA is severely stenosed or occluded proximal to the branch to the right ventricle. However, inferior wall MI may also occur secondary to occlusion of the circumflex coronary artery (CA). In the latter situation RVI is not expected. In the present study coronary angiograms of patients with a documented inferior wall MI are correlated with data from the unipolar right precordial lead V_4R at the time of the acute MI. The purpose of study was to assess the value of V_4R in identifying both the level and site of right coronary artery (RCA) or CA occlusion and the presence or absence of RVI.

Patients and methods

The patients including had to fulfill the following entrance criteria:

I. admission to the coronary care unit within 10 hours after the onset of chest pain
II. no history or documentation of a previous inferior wall MI
III. presence of an acute inferior wall MI. The diagnosis of MI was based on clinical history, characteristic enzyme pattern and evolutionary ECG changes with the appearance of pathologic Q-waves in the inferior leads
IV. no reinfarction before this catheterisation
V. young males (40 years) or recurrence of chest pain in spite of medical treatment.

Eighty-eight patients fulfilled these criteria. Ages ranged from 33-71 (mean 55) years. In 79 patients there was no documented myocardial infarction in the past and 9 had already suffered from an anterior wall MI. A standard 12-lead ECG and a right sided chest lead V_4R was taken on admission and afterwards every 8 hours during 3 days. ECG's were interpreted by two cardiologists and ST- segment elevation $\geqq$ 1 mm in V_4R on admission was judged to be a positive sign for RVI (2-6). Venous blood samples for serum glutamic oxaloacetic transaminase and creatinephosphokinase were drawn on admission and every 8 hours during 3 days and thereafter daily for 2 days. A coronary angiograms was performed 2 to 26 weeks (mean 10) after the acute MI. Reasons for catheterisation were: MI in young males ($\leqq$ 40 years) and recurrence of chest pain in spite of medical treatment in the remaining patients.

A left ventriculography was performed in the right and left anterior oblique projection. Right and left coronary artery angiography was performed using multiple views, including angulated projections. A $\geqq$ 50% reduction in the luminal diameter of a coronary artery was considered significant. The coronary angiograms were judged by two cardiologists without knowledge of the electrocardiographic findings. Differences in opinion were resolved by discussion. If the RCA and the CA were both diseased, the artery with the subtotal luminal narrowing or a total obstruction was believed to be the cause of the MI.

Results (Table 1)

On admission 36 out of 88 patients (43%) had ST-segment elevation $\geqq$ 1 mm in lead V_4R. Two patients of this group showed initially signs of right sided heart failure.

Table 1

Presence or absence of ST-segment elevation in $V_4R \geqq 1$ mm and the site and degree of lesions in the coronary arteries of the 88 patients studied

No.	Age	V_4R	RCA	LAD	CA		
1	61	N	**95**	75			
2	43	N			**75**		
3	52	N		60	**75**		
4	51	pos	**100**	100		*	
5	70	pos	**95**				
6	62	N			**100**		
7	49	pos	**100** prox	70			
8	42	pos	**100** prox	75			
9	67	pos	**100** prox	90			
10	54	N	**75**	50			
11	48	N	**95**	95	75	*	
12	66	pos	**95**	80	50		
13	59	pos	**100** prox	90			
14	48	N	**100**	75			
15	57	N	**90**	80	80		
16	54	pos	**100**	100			
17	52	pos	**100**	100	80	*	
18	41	pos	**100** prox	75	90		
19	47	N	**100**	70			
20	62	pos	**100**	80	80		
21	63	N			**99**		
22	59	pos	**100** prox	50			
23	53	pos	**90** prox	70			
24	42	N	**95**		90		
25	71	N	75	75	**90**		
26	33	N		90	**99**		
27	60	N	**95**				
28	67	N	100	50	99	*	0
29	54	N	**100**				
30	41	N		60	**100**		
31	38	pos	**100** prox				
32	62	N	**100**	70			
33	55	N	**75**	80			
34	42	pos	**100** prox	90	80		

No.	Age	V_4R	RCA	LAD	CA		
35	62	N		60	**100**		
36	62	pos	**90** prox				
37	43	N	**100**	75	60		
38	62	N	**100**	100	80	*	
39	57	N		50	**90**		
40	56	N	70	60	**95**		
41	71	N	**95**				
42	53	N	100	60	100	*	0
43	33	pos	**100** prox	60			
44	54	N	**100**	75	75		
45	39	N		80	**85**		
46	45	N	**80**				
47	39	pos	**100** prox				
48	62	pos	75	95	75	*	0
49	50	pos	**100** prox	80	80		
50	46	N			**95**		
51	46	pos	**100** prox	90			
52	60	N		70	**80**		
53	38	N	**100**				
54	59	N	80	95	**100**		
55	54	N			**100**		
56	66	pos	**90** prox	95			
57	59	N	80	70	**100**		
58	60	N	**100**	80			
59	44	pos	80	60	80	*	0
60	69	N		50	**95**		
61	63	N	**100**	60	70		
62	57	N			**100**		
63	53	pos	**100** prox	60			
64	62	N	**95**	70	50		
65	56	N	**100**	90	70		
66	60	pos	**100** prox	70	80		
67	40	N		80	**100**		
68	64	N	**100**	85			
69	54	N			**100**		
70	65	N	**100**				
71	64	N	80	75	**95**		
72	59	pos	**100** prox	80			
73	54	pos	**100** prox	80	75		

No.	Age	V_4R	RCA	LAD	CA
74	52	N	80	75	**100**
75	60	pos	**100** prox		
76	59	N	**100**	90	80
77	47	pos	**100** prox		
78	51	N	60	75	**100**
79	70	pos	**100** prox	80	
80	55	N	**100**	90	90
81	53	pos	**100** prox	60	
82	53	pos	**100**	80	
83	58	N		60	**100**
84	65	pos	**100** prox	70	
85	63	pos	**100** prox		
86	66	N	**100**	60	80
87	54	pos	**100** prox	80	60
88	50	pos	**100** prox		

* Previous anterior wall infarction.

0 Impossible to judge which stenosis causes the infarction.

The bold-faced numbers indicate the coronary artery judged to be the cause of the myocardial infarction.

Abbreviations: prox = occlusion proximal to the first branch to the right ventricle; RCA = right coronary artery; LAD = left anterior descending coronary artery; CA = circumflex coronary artery; N = no elevation in V_4R; pos = ST elevation $\geqq$ 1 mm in V_4R.

Catheterisation data:

Twenty-one patients had single vessel disease, in 14 patients only the RCA and in 7 cases only the CA was affected. Two vessel disease was found in 35 patients, in 24 cases the RCA and the left anterior descendens (LAD) and in 10 the CA and the LAD. Only 1 patient had simultaneous significant luminal narrowing of the RCA and the CA. In 32 patients three vessel disease was found. In 4 patients of the group with three vessel disease the stenosis in the RCA as well as in the CA was so severe, that it was impossible to judge which stenosis had caused the MI. These 4 patients are therefore not included in the correlation data of catheterisation and electrocardiographic recordings. This leaves 84 patients for analysis. In 68% (57 out of 84 patients) a complete occlusion of at least one coronary artery was present. In 20 patients (24%) a stenosis between 90 and 95% was found and in 7 patients (8%) a narrowing between 75 and 90% was found and considered to be the cause for the MI. No patient in this study was found to have normal coronary arteries.

For correlation with the ECG findings we divided the site of coronary artery stenosis in patients in whom it was possible to judge which coronary artery has caused the inferior wall MI (84 patients), in three groups:
a. stenosis proximal to the first branch to the right ventricle;
b. distal to the branch of the right ventricle; or
c. in the CA.
On comparing the electrocardiographic findings of lead V_4R with the catheterisation findings, we found that none of the 21 patients with CA stenosis had ST-segment elevation in lead V_4R. All 27 patients in whom the stenosis was located above the first branch to the right ventricle, had ST-segment elevation of more than 1 mm in lead V_4R (Table 2). From the group of 36 patients in whom the stenosis was located below the first branch to the right ventricle 7 showed ST-segment elevation in V_4R, while that was absent in 29 patients. Of these 7 patients, 3 had three vessel disease, 3 two vessel disease and 1 single vessel disease.

Table 2

Presence and absence of ST-segment elevation in lead $V_4R \geqq 1$ mm and site of occlusion in coronary artery

	V_4R +	V_4R −
Occlusion of the RCA above the first branch to the right ventricle N = 27	27	0
Occlusion of the RCA distal to the first branch to the right ventricle N = 36	7	29
Occlusion of the circumflex coronary artery	0	21

Abbreviations: RCA = right coronary artery

The sensitivity of ST-segment elevation in lead V_4R to predict an occlusion above the first branch to the right ventricle is 100%, the specificity in this group is 87%, while the predictive accuracy is 92%.

Discussion

Several studies have demonstrated the value of ST-segment elevation in lead V_4R for diagnosing RVI in patients with inferior wall MI (2-6). Involvement of the right ventricle has been demonstrated in these patients by pathological (2), scintigraphic (3, 4, 5) and hemodynamic studies (6). Inferior wall MI can occur in relation to oc-

clusion of the RCA or CA, but blood supply to the free wall of the right ventricle occurs in more than 90% of patients by way of a branch of the RCA (8, 9). One would therefore expect that the electrocardiographic evidence for RVI (ST-elevation in lead V_4R) would also have a high sensitivity and specificity to identify occlusion of the RCA. The results of our study (Table 2) show that ST-segment elevation of $\geqq$ 1 mm in lead V_4R in patients with acute inferior MI has a sensitivity of 100% and a specificity of 87% for a stenosis proximal to the branch of the right ventricle of the RCA, the predictive accuracy is 92%. Seven out of 36 patients with a distal occlusion of the RCA showed ST-segment elevation of $\geqq$ 1 mm in lead V_4R. In patients with inferior wall MI and occlusion distal to the first branch to the right ventricle of the RCA, ST-segment elevation in lead V_4R could indicate involvement of the right ventricle by occlusion of the 2nd and 3rd right ventricular branch of the RCA to the right ventricle. Absence of ST-segment elevation in lead V_4R in patients with acute inferior wall MI is, however, of no help to identify RCA or CA involvement, but excludes proximal occlusion of the RCA.

Our study was retrospective and the patient population was selected. In our department we do not routinely catheterize all patients after MI, but only those patients presenting a particular diagnostic, prognostic or therapeutic problem. Our prevalences of total or subtotal occlusion of the coronary arteries was however similar to the prevalences, found in unselected patients catheterized 1 month post-MI (10).

Clinical implications:

Our results may have clinical implications for patients with an acute MI. Patients with unstable angina pectoris or acute MI are presently being more aggressively treated than in the past. Several centers are performing studies on the value of intracoronary thrombolysis with streptokinase, balloon dilatation of coronary arteries and bypass surgery. Knowledge about the site of obstruction in the coronary arteries can expedite some of these procedures. The simple recording of lead V_4R in patients with acute inferior wall MI can give rapid information about occlusion of the RCA (ST elevation in lead $V_4R \geqq 1$ mm) and help to exclude proximal occlusion. As previously discussed (3, 11) ST-segment elevation disappeares rapidly in patients with inferior wall MI, 50% disappeared within within 10 hours (3). Therefore, electrocardiographic recordings should be made as soon as possible after admission. In our department recording of right precordial leads in patients admitted with inferior MI has become a routine procedure.

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Chapter 5

Intracoronary thrombolysis for acute myocardial infarction late after bypass surgery: value of lead V_4R

by

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Published in the American Journal of Cardiology 1984, 53: 637-639.

Case report

Lysis of intracoronary thrombi in the acute stage of myocardial infarction (MI) can be effectively achieved by administration of thrombolytic agents in 60-85% of patients when it is given within 6 hours after the onset of chest pain (1-3). We recently studied a patient admitted because of an acute MI in whom coronary bypass surgery had been performed 5 years earlier. The recording of lead V_4R was of great help in recognizing the occluded vessels.

A 60 year old man was admitted because of chest pain not reacting to nitroglycerin and nifedipine sublingually. He had had angina for 10 years, with bypass surgery at age 55. The catheterisation findings before bypass surgery showed a proximal occlusion in a dominant right coronary artery which was filled retrogradely by the left anterior descending (LAD) artery. The LAD artery showed a 85% diameter reduction just after the first diagonal branch, there was a 75% stenosis at the origin of the first obtuse marginal (OM) branch of the circumflex coronary artery (CA). A small infero-apical aneurysm was found on left ventricular angiography. Coronary bypass graft surgery was performed with 1 graft to the right coronary artery, 1 to the LAD and 1 to the first OM branch. The postoperative period was uneventful and following operation the patient was free of complaints until the present admission.

Blood pressure was 130/80 mmHg, the pulse rate 95 beats/minute. A fourth heart sound was present. The electrocardiogram on admission showed signs of an acute infero-posterior wall MI (Fig. 1). Lead V_4R showed ST-segment elevation of 1 mm indicating ischemia of the right ventricle (4, 5).

Since the patient was admitted 1 hour after the onset of chest pain streptokinase was started 100,000 IU intravenously. Because of the ST-segment elevation in lead V_4R we suspected ischemia of the right ventricle and therefore problems in the graft to the right coronary artery and not in the one to the OM branch of the CA. Using the Judkins technique a catheter was introduced in the right coronary artery bypass graft. This bypass was found to be proximally occluded (Fig. 2A). After 200,000 IU of streptokinase the artery opened up (Fig. 2B) and immediately the pain and ST-segment elevation in II, III and AVF and in V_4R disappeared (Fig. 3). We then proceeded with the catheterization. The 2 other bypasses were patent. The left coronary artery showed the same abnormalities as 5 years earlier.

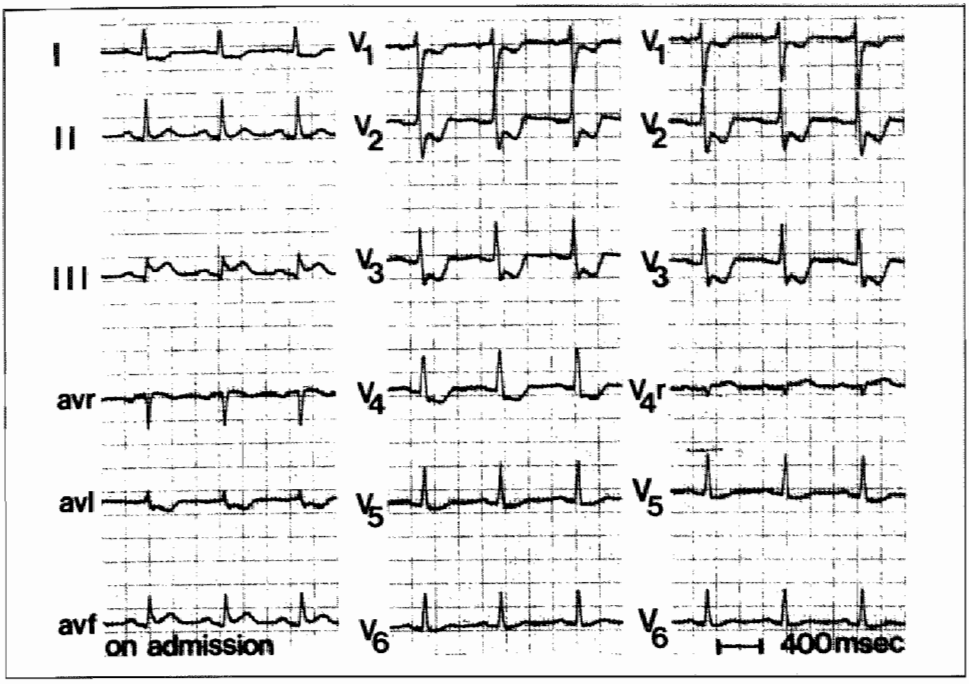

Figure 1 The 12 lead electrocardiogram shows an acute infero-posterior wall myocardial infarction. ST-segment elevation of 1 mm is present in lead V_4R.

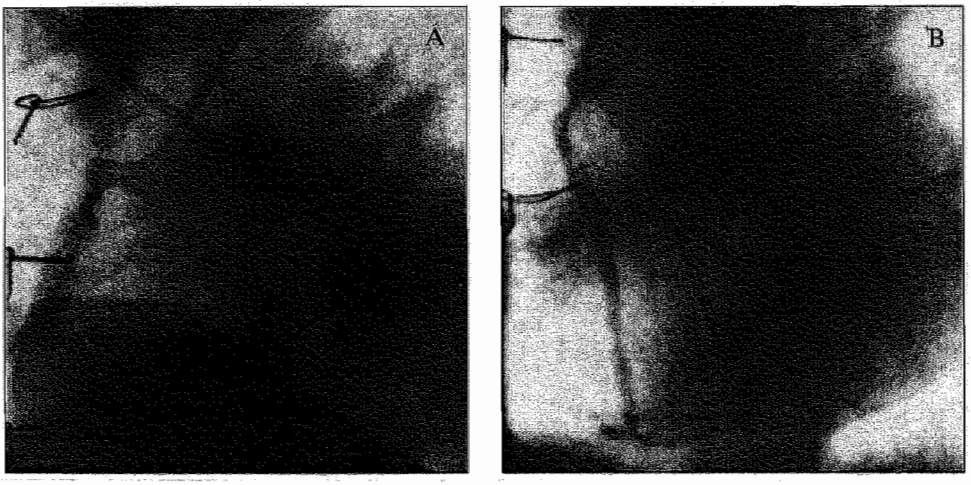

Figure 2. A. Before streptokinase angiography of the bypass to the right coronary artery shows complete occlusion.

B. After streptokinase distal filling of the bypass is present with visualization of the right coronary artery. An 80% stenosis is visible in the bypass graft.

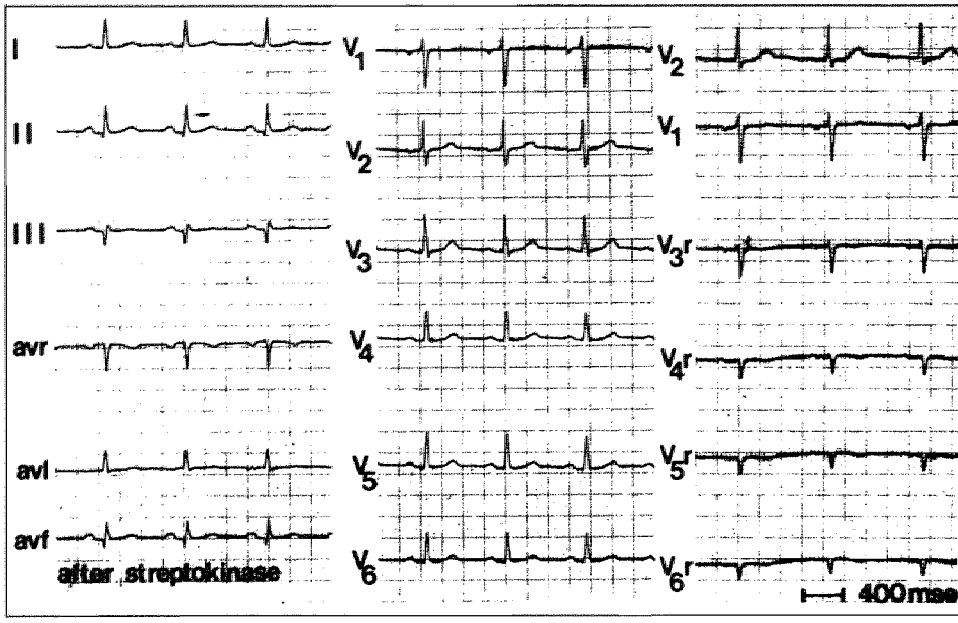

Figure 3 Electrocardiogram recorded after streptokinase infusion. The signs of the acute infero-posterior wall myocardial infarction have disappeared. ST-segment elevation in lead V_4R is no longer present.

The ECG returned to the one during his last out-patient clinic visit. There was only a slight enzyme rise. The maximal creatine phosphokinase value became 360 (normal value $\leqq$ 240) IU.

The recording of lead V_4R can be of importance in deciding which coronary artery system should be investigated first for streptokinase therapy. Five years ago in our patient the right coronary artery was occluded. Thus, we believed that the bypass on the right coronary artery had to be occluded and the artery was immediately infused with streptokinase. We and other investigators (4, 5) have shown that in patients with an acute inferior wall MI ST elevation of $\geqq$ 1 mm in lead V_4R very strongly suggests involvement of the right ventricle with proximal occlusion of the right coronary artery. In almost 90% the blood supply to the right ventricle is coming from the right coronary artery and in 10% from the LAD (6). When our patient presented with an acute inferior wall MI with the information of having bypass grafts to a proximally occluded right coronary artery and a proximally narrowed OM branch of the CA the ST-segment elevation in lead V_4R indicated to us a perfusion problem in the right coronary artery. Therefore, we immediately directed our attention to the bypass graft on the right coronary artery which indeed

was found to be occluded. In the setting of an acute inferior wall MI, when no ST-segment elevation is seen in the right precordial lead V_4R, the probability of an occlusion of the CA or the right coronary artery is almost equal. Non-invasive methods should be found to make it possible to distinguish between a perfusion problem in the right from one in the CA in these patients.

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Chapter 6

Value of lead V_4R in exercise testing to predict proximal stenosis of the right coronary artery

by

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Submitted for publication: American Journal of Cardiology

Summary

To assess the value of lead V_4R during exercise testing to predict proximal stenosis of the right coronary artery 107 patients were studied. In all patients a Bruce exercise test with the simultaneous recording of leads I, II, V_4R, V_1, V_4 and V_6 was followed by coronary angiography. Apart from registering ST-segment changes in the conventional leads, all patients were classified according to absence or presence of an ST-segment deviation of $\geqq$ 1 mm in lead V_4R. Seventy-nine of the 107 patients were studied because of inadequate control of angina pectoris. Seven patients had suffered from a myocardial infarction under age 40. Twenty-one patients were analysed because of severe cardiac arrhythmias. In the 46 patients who previously had a myocardial infarction, the infarct location was inferior in 28 and anterior in 18. Seven of the 14 patients without myocardial infarction and a significant stenosis proximal in the right coronary artery showed during exercise ST-segment deviation $\geqq$ 1 mm in lead V_4R. This was also observed in 11 of 18 patients with an old inferior wall infarction and a proximal occlusion of the right coronary artery. None of the 53 patients without significant stenosis in the right coronary artery showed exercise related ST-segment changes in lead V_4R. Exercise related ST-segment deviation in lead V_4R had a sensitivity of 56%, a specificity of 96% and a predictive accuracy of 84% in recognizing a proximal stenosis in the right coronary artery. Our observations indicate that the recording of lead V_4R during exercise is of value to predict or exclude proximal stenosis in the right coronary artery.

In 1950 Master (1) popularized the 2 step exercise test. In 1957 Bruce and Hornsten (2) introduced high-performance treadmill exercise testing for the detection and evaluation of coronary artery disease. In the last decade, several reports (3, 4) showed that the value of the exercise test can be improved by adding the analysis of other variables to the electrocardiographic changes during exercise. Further improvement in the value of electrocardiographic detection of ischaemia can be achieved by using multiple electrocardiographic leads (5). The presence of ST-segment elevation in lead V_4R in an acute inferior wall myocardial infarction has been shown to be a sensitive and specific sign of right ventricular involvement (6-10). ST-segment elevation in V_4R is almost exclusively seen during obstruction of the right coronary artery proximal to the first branch to the right ventricle (11). The purpose of this study was to assess the value of changes in lead V_4R during exercise stress testing to predict proximal stenosis in the right coronary artery.

Methods:

Patient population

One hundred and seven consecutive patients admitted for coronary angiography were studied. In 79 patients chest pain with inadequate reaction to anti-anginal medication was present. Seven patients were below the age of 40 and had suffered a myocardial infarction. Twenty-one patients were analysed because of severe rhythm disturbances. There were 18 women and 89 men. Age ranged between 27 and 72 (mean 54 ± 10) years. All patients with chest pain were receiving long acting nitroglycerine, a calcium channel blocking agent and a B-blocking agent. No patient had signs of the Wolff-Parkinson-White syndrome, left ventricular hypertrophy or bundle branch block. Forty-six patients had sustained a previous myocardial infarction, 18 anterior and 28 an inferior wall myocardial infarction.

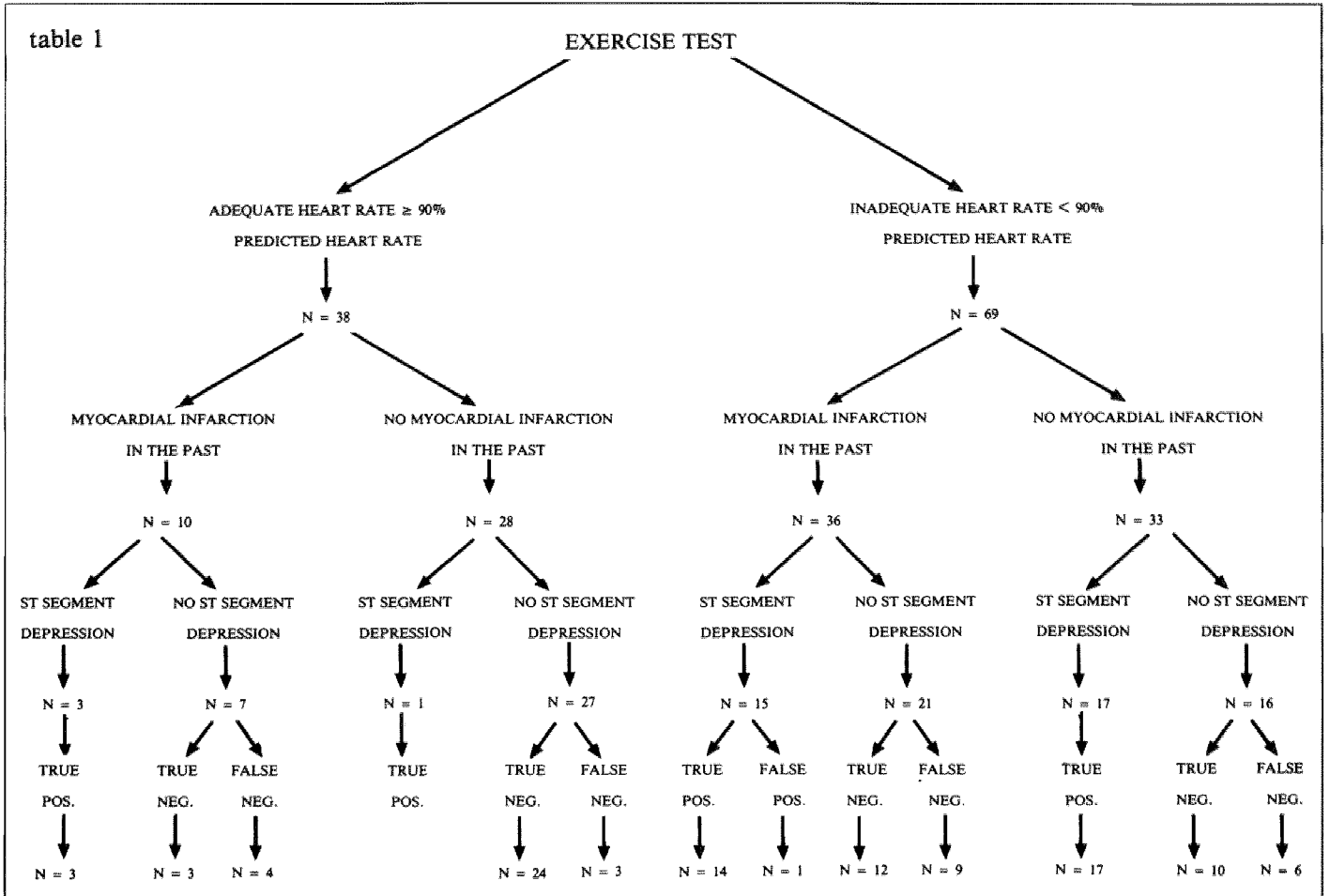
Exercise testing

None of the patients had evidence of impending or healing myocardial infarction or an acute general illness. Six electrocardiographic leads (I, II, V_4R, V_1, V_4 and V_6) were recorded simultaneously. V_4R was placed on the right chest wall in the midclavicular line in the fifth intercostal space. The arm electrodes were applied in the far lateral aspects of the infraclavicular fossae and a standard electrocardiogram was recorded at rest in the recumbent and standing position and interpreted for contraindications before starting exercise. At the end of each 3-minute exercise stage, the 6 electrocardiographic leads, as mentioned above were recorded and every 60 sec. thereafter for at least 10 minutes or until resolution of any exercise-induced abnormalities. The treadmill protocol of Bruce and Hornsten (2) was used and patients were allowed to touch the front handrail but not to lean or pull on it. Exercise was stopped because of fatigue, shortness of breath, leg discomfort or chest pain with/without ST-segment depression. Achieving the maximal predicted heart rate according to age was no reason to stop exercising.

An ST-T abnormality suggestive of ischemia was defined as ST-T depression of at least 1 mm and lasting at least 80 ms after the J-point. In patients with ST-segment deviation in the resting electrocardiogram an additional 1 mm of ST-T depression was required. In lead V_4R the same approach was used, but in addition, also ST-segment elevation of more than 1 mm lasting at least 80 ms after the J-point was noticed. The test was considered negative if there was no ST-segment deviation $\geqq$ 1 mm.

Only when 90% of the age predicted maximal heart rate was achieved the test was considered adequate. Exercise tests were judged independently by two cardiologists and disagreement was resolved by consensus.

table 1


EXERCISE TEST
ADEQUATE HEART RATE ≥ 90% PREDICTED HEART RATE
INADEQUATE HEART RATE < 90% PREDICTED HEART RATE
N = 38
N = 69
MYOCARDIAL INFARCTION IN THE PAST
NO MYOCARDIAL INFARCTION IN THE PAST
MYOCARDIAL INFARCTION IN THE PAST
NO MYOCARDIAL INFARCTION IN THE PAST
N = 10
N = 28
N = 36
N = 33
ST SEGMENT DEPRESSION
NO ST SEGMENT DEPRESSION
ST SEGMENT DEPRESSION
NO ST SEGMENT DEPRESSION
ST SEGMENT DEPRESSION
NO ST SEGMENT DEPRESSION
ST SEGMENT DEPRESSION
NO ST SEGMENT DEPRESSION
N = 3
N = 7
N = 1
N = 27
N = 15
N = 21
N = 17
N = 16
TRUE POS.
TRUE NEG.
FALSE NEG.
TRUE POS.
TRUE NEG.
FALSE NEG.
TRUE POS.
FALSE POS.
TRUE NEG.
FALSE NEG.
TRUE POS.
TRUE NEG.
FALSE NEG.
N = 3
N = 3
N = 4
N = 1
N = 24
N = 3
N = 14
N = 1
N = 12
N = 9
N = 17
N = 10
N = 6

Coronary angiography

All patients had selective coronary angiography by the Judkins technique (12) within one week after the performance of the exercise test. The coronary arteries were viewed and filmed in multiple and angulated projections. Angiograms were independently interpreted by two cardiologists unaware of the results of the exercise test. Disagreement in judgement was resolved by consensus. A stenosis of more than 50% was considered to be significant. It was also noticed if a stenosis in the right coronary artery was located above or below the first branch to the right ventricle.

Results (Table I)

Thirty-eight patients (35%) achieved at least 90% of the predictive heart rate, 10 of them had previously suffered from a myocardial infarction. Four patients showed ST-segment depression $\geqq$ 1 mm in one or more leads, 3 of them had suffered from a myocardial infarction in the past. Of the group of 69 patients who did not achieve 90% of the predictive heart rate, 32 patients showed ST-segment depression of more than 1 mm in one or more leads. An old myocardial infarction was present in 15 of these 32 patients. In total 36 patients showed diagnostic ST-segment depression with 18 patients previously having suffered from a myocardial infarction.

Catheterisation data

Thirty-four patients had normal coronary arteries, 22 patients had single vessel disease; 16 of them had suffered from a myocardial infarction in the past; 25 patients had two vessel disease and 26 patients had three vessel disease.

Correlation between the conventional exercise ECG and catheterisation data

In the group of patients with single vessel disease and an old myocardial infarction a negative exercise test was considered to be true negative.

In the group of patients with an old myocardial infarction and two or three vessel disease a positive test was considered to be true positive if a significant stenosis was present in a coronary artery other than the infarct vessel. A true positive exercise test was found in 35 patients; a false negative test in 22 patients and a true negative test in 49 patients. One patient had a false positive exercise test.

In the group of 38 patients with an adequate exercise test 4 patients had a ST-

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Table 1 Exercise test

The exercise tests are divided in groups of patients with or without achieving the predicted heart rate and with or without an old myocardial infarction and the numbers of true positive, true negative, false negative and false positive are given.

segment depression of more than 1 mm in the presence of significant stenoses. Seven patients of this group had no ST-segment depression, while the coronary angiogram showed significant stenoses, 4 of them had suffered from an old myocardial infarction and had at least two significant stenoses.
Twenty-four patients with an adequate exercise test had normal coronary arteries and had no ST-segment depression of more than 1 mm. Three patients had an old infarction and single vessel disease and had no ST-segment depression. Of the 69 patients with an inadequate exercise test 32 patients showed ST-segment depression and 31 had significant stenoses. Fourteen of these patients had suffered from an infarction in the past. Fifteen of the 69 patients with an inadequate exercise test had no ST-segment deviation of more than 1 mm, while significant stenoses were found in the angiogram. Nine patients of this group had suffered from an infarction in the past and had at least two vessel disease.
In 22 of the 69 patients no ST-segment depression was seen while in 10 patients normal coronary arteries were found. The other 12 patients had an old infarction and single vessel disease.
All 22 were considered to be true negative. One patient with an old inferior wall myocardial infarction and single vessel disease of the right coronary artery and a positive exercise test was considered false positive.
So, based on observations on the conventional ECG leads during exercise, the sensitivity of ST-segment depression $\geqq$ 1 mm in all 107 patients to predict significant stenoses is 60% while the predictive accuracy is 79%.
The sensitivity in the group of patients with an adequate exercise test is 36%, while the predictive accuracy is 82%. For the group of patients without an inadequate exercise test the sensitivity is 67% and the predictive accuracy 78%.

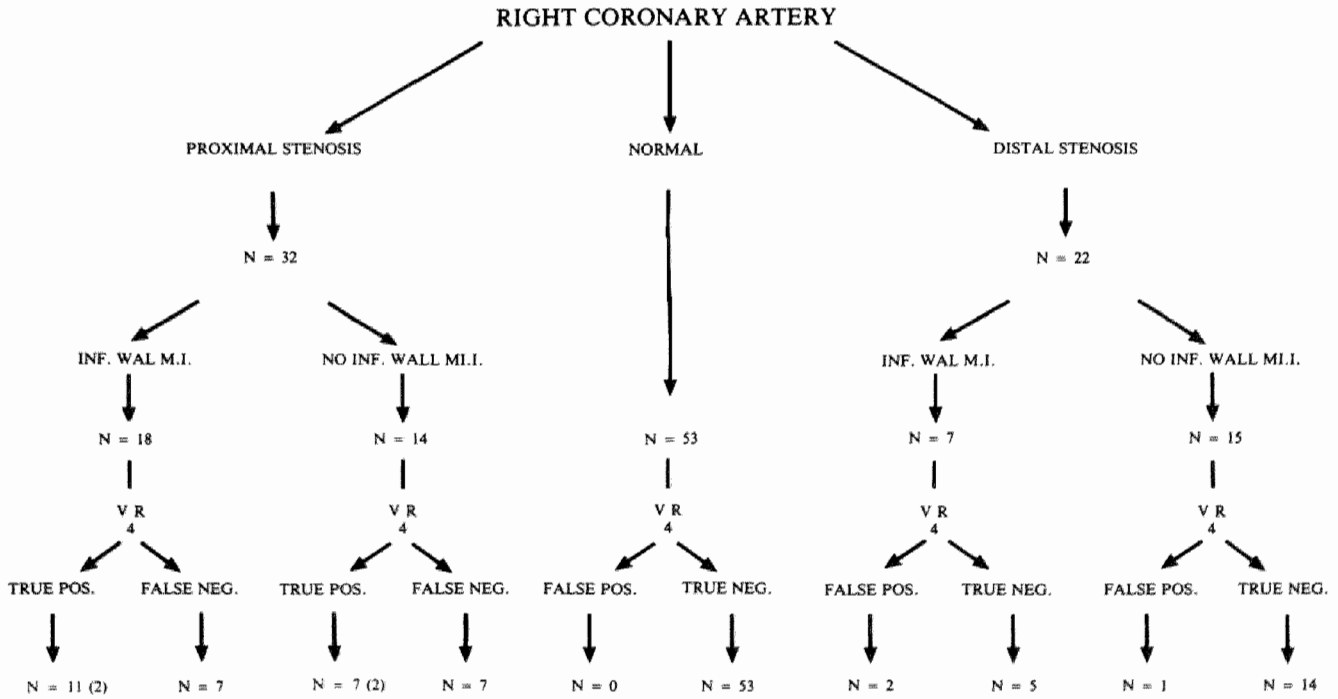
Findings in lead V_4R

Eighty-six patients had no ST-segment elevation or depression in V_4R of more than 1 mm. In 17 patients ST-segment elevation in V_4R of more than 1 mm was found. In 4 patients we found a significant depression in lead V_4R.

Table 2 Correlation between the catheterisation data and V_4R

The coronary angiographic data of the right coronary artery are given. The groups of significant stenoses in the right coronary artery are then divided in patients with or without an old myocardial infarction on the ECG. The end of the table listed the numbers of true positive, true negative, false positive and false negative deviation in V_4R to predict proximal stenosis in the right coronary artery. The numbers in parenthesis are the numbers of ST-segment depression in $V_4R \geqq 1$ mm.

table 2


RIGHT CORONARY ARTERY
PROXIMAL STENOSIS
NORMAL
DISTAL STENOSIS
N = 32
N = 22
INF. WAL M.I.
NO INF. WALL MI.I.
INF. WAL M.I.
NO INF. WALL MI.I.
N = 18
N = 14
N = 53
N = 7
N = 15
V R 4
V R 4
V R 4
V R 4
V R 4
TRUE POS.
FALSE NEG.
TRUE POS.
FALSE NEG.
FALSE POS.
TRUE NEG.
FALSE POS.
TRUE NEG.
FALSE POS.
TRUE NEG.
N = 11 (2)
N = 7
N = 7 (2)
N = 7
N = 0
N = 53
N = 2
N = 5
N = 1
N = 14

Catheterisation data of the right coronary artery

Thirty-two patients had at least a narrowing of more than 50% in the right coronary artery proximal to the first branch to the right ventricle.

Of these patients with a proximal significant stenosis of the right coronary artery 18 patients had suffered from an inferior wall myocardial infarction in the past. Twenty-two patients had a significant stenosis distal to the first branch to the right ventricle, 7 of them had suffered from an inferior wall myocardial infarction in the past. In 3 patients an occlusion in the circumflex artery was judged to be the cause of the inferior wall myocardial infarction. Single vessel disease of the right coronary artery was found in 13 patients. Ten had a significant stenosis proximal to the first branch to the right ventricle. Of these 13 patients 10 had an inferior wall infarction in the past. Three with an occlusion distal to first branch to the right ventricle and 7 with an occlusion proximal to the first branch to the right ventricle.

In 54 patients a significant stenosis was present in the right coronary artery, 32 patients had a proximal stenosis of whom 18 had suffered from an inferior wall myocardial infarction.

In this group of 18 patients with an old inferior wall myocardial infarction caused by an occlusion proximal to the first branch of the right ventricle 11 patients showed ST-segment deviation $\geqq$ 1 mm in lead V_4R. In the 14 patients with a proximal obstruction in the right coronary artery without an inferior wall myocardial infarction 7 times ST-segment deviation $\geqq$ 1 mm was noticed in lead V_4R.

In 22 patients a distal obstruction was present in the right coronary artery, 7 of them had suffered from an old inferior wall myocardial infarction and 2 of them had ST-segment elevation $\geqq$ 1 mm in lead V_4R. Only 1 patient in the group of 15 patients without an old inferior myocardial infarction and a significant stenosis distal to the first branch to the right ventricle showed ST-segment elevation in $V_4R \geqq 1$ mm. None of the 53 patients without a significant stenosis in the right cornary artery showed ST-segment deviation $\geqq$ 1 mm in lead V_4R.

If we calculate the sensitivity, specificity and predictive accuracy of ST-segment elevation/depression in V_4R we come to a sensitivity of 56%, a specificity of 96% and a predictive accuracy of 84%.

Discussion

In spite of the fact that the exercise test was performed in a group of patients without discontinuation of anti-anginal treatment and included 21 patients with severe rhythm disturbances results of sensitivity and predictive accuracy of recognition of coronary heart disease using conventional ECG leads are comparable with results of other investigations (13). The specificity of diagnosing ischemic cardiac disease was very high (84%). Our group of 107 patients consisted of 18 women. Eight of

them had a true negative exercise test, 4 a false negative exercise test and 6 a true positive exercise test. We have no explanation for the absence of false positive exercise test in our female patients.

To accept a negative exercise test in patients with an old myocardial infarction and single vessel disease as true negative can be a point of discussion. As mentioned in the method section in the conventional ECG leads only ST-segment *depression* was accepted as suggestive for ischemia. Therefore, ST-segment *elevation* which may occur in the leads recording from the area of infarction in absence of additional ischemia was excluded. Ideally, to solve this problem a thallium exercise test would have been of value. An exercise test with a defect which remains unchanged in a patient with an old myocardial infarction and single vessel disease would support the diagnosis of a true negative exercise test. However, no thallium exercise tests were performed in this group of patients.

For the evaluation of the value of V_4R to detect ischemia of the right ventricle thallium scintigraphy is of limited value, because the right ventricle is shown in only a low percentage of the redistribution studies.

Until now we do not have a good explanation for the elevation of lead V_4R in patients having an acute inferior wall myocardial infarction with a right ventricular infarction. Is the elevation caused by involvement of the posterior part of the septum or by ischemia of the free wall of the right ventricle? It is of importance that none of the patients with a normal right coronary artery had ST-segment deviation in V_4R, also when a significant stenosis in the circumflex coronary artery with or without myocardial infarction was present. This indicates that ST-segment deviation in V_4R is not caused by reciprocal ST-segment changes from perfusion abnormalities in the posterior wall.

Twenty-one of our patients showed ST deviation $\geqq$ 1 mm in lead V_4R, with only 7 of 14 patients having a significant stenosis in the right coronary artery proximal to the first branch to the right ventricle in the absence of an old inferior wall myocardial infarction. In the group of 18 patients with an old inferior wall myocardial infarction caused by a proximal occlusion of the right coronary artery 11 patients showed ST-segment deviation. These patients can be considered as being false positive but it is also possible that they are true positive with the right ventricle still having jeopardized myocardium. To solve this problem one might be tempted to determine the ejection fraction of the right ventricle during exercise. In 1979 Berger et al (14), however, showed that the right ventricular function during exercise is not primarily dependent on the presence or absence of a proximal right coronary artery stenosis. Nevertheless, our data show that recording lead V_4R during exercise is of help to identify patients with a proximal stenosis in the right coronary artery with or without an old inferior myocardial infarction.

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Chapter 7

Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrio ventricular nodal conduction disturbances

by

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Published in the American Heart Journal 1984, 107: 1183-1186. In part presented at the 31 Annual Scientific Meeting of the American College of Cardiology, Atlanta 1982.

Summary

In 67 consecutive patients with acute inferior wall myocardial infarction, 99mTechnetium pyrophosphate scintigraphy was performed 36 to 72 hours after the onset of chest pain to detect right ventricular involvement. All patients were continuously monitored during at least 3 days to detect rhythm and conduction disturbances.
In 29 patients right ventricular involvement was diagnosed by scintigraphy. None of these 29 patients showed clinical signs of right sided heart failure. Fourteen of the 19 patients showing atrioventricular (AV) nodal conduction disturbances in the setting of an inferior MI also had involvement of the right ventricle (RV). Therefore, the incidence of high degree AV nodal block in patients with RV involvement (14 of 29 patients) was 48% compared to only 13% (5 of 38) in patients with an inferior MI without RV involvement.

In patients admitted because of acute MI, high degree atrioventricular (AV) block has been reported to occur in 12 to 25% of cases (1-3). It has been shown that the incidence of high degree AV block is 3 times higher in patients with an inferior as compared to patients with an anterior wall infarction (4). This implies that the incidence of high degree AV block in inferior MI is approximately 27% (18-36%) (5). The occurrence of AV nodal block is usually explained by the fact that the blood supply to the AV node depends in 90% of patients on the right coronary artery (RCA) (6). One might therefore, expect a very high incidence of AV nodal block in patients in whom an inferior MI is accompanied by RV involvement because occlusion of the RCA should then be proximal to the branch to the AV node.
To study this hypothesis data from 67 consecutive patients, admitted to our coronary care unit, with an inferior MI were retrospectively analysed.

Methods

Patients.

Sixty-seven consecutive patients with an inferior MI were studied retrospectively. Fifty-six were male and 11 female. Ages ranged from 39 to 80 (mean 57 ± 9.4) years. Four patients had suffered from a documented myocardial infarction in the past, 2 previously had had an anterior and 2 an inferior wall myocardial infarction. The diagnosis of inferior MI was based on the clinical history, characteristic rise in CPK and SGOT values and appearance of new pathologic Q waves in the inferior leads.

99mTechnetium pyrophosphate scintigraphy.

In each patient 99mTechnetium pyrophosphate scintigraphy was performed 36 to 72 hours after the onset of chest pain. Sixty to 90 minutes after the injection of 15-20 mC of 99mTechnetium pyrophosphate, 3 views were recorded with a general all purpose parallel hole collimator: the anterior, left lateral and 45° left anterior oblique (LAO). Each view contained at least 600,000 counts.

After the last view, (the 45° LAO-projection) was recorded, a small bolus of 99mTechnetium was injected without moving patient or collimator. At the same time a dynamic flow study was performed, using frames of 1 second to visualize separately the RV and the left ventricle. A region of interest was placed where the RV was visualized. By superimposing this area of the RV on the previously obtained 45° LAO view RV involvement was identified. An Ohio Nuclear Sigma 420 mobile gamma camera with a general all purpose parallel hole collimator interfaced to a MCS-560 mobile computer system and a Philips gamma camera with a general all purpose parallel hole collimator interfaced to a PDS computer system were used for all studies. The radionuclide data were analysed by 2 independent observers without knowledge of the clinical data. The 99mTechnetium pyrophosphate scintigraphy was judged to be positive when there was definite myocardial uptake. RV involvement was considered to be present if definite myocardial uptake was seen in the region of interest of the RV (Fig. 1).

ECG monitoring.

A 12-lead ECG plus four additional right precordial leads (V_3R, V_4R, V_5R and V_6R) were recorded on admission and routinely every 8 hours during the next three days. Leads V_3R, V_4R, V_5R and V_6R are the mirror images of lead V_3, V_4, V_5 and V_6 on the right chest wall. At the same time blood samples were drawn to determine CPK and SGOT values.

All patients had continuous ECG monitoring for at least 3 days to detect rhythm and conduction disturbances. AV block was thought to be located at the AV nodal level when second degree (2:1 or Wenckebach) block developed in the presence of a narrow QRS-complex. Third degree AV block was considered to be located at the AV nodal level when it was associated with an escape rhythm showing a QRS-complex similar to the one recorded during the conducted supraventricular rhythm. No patient had right or left bundle branch block during MI. When conduction disturbances were noted complete electrocardiograms including the additional right precordial leads were recorded immediately (Fig. 2).

In none of our patients was invasive hemodynamic monitoring performed, because physical examination revealed no signs of right sided heart dysfunction.

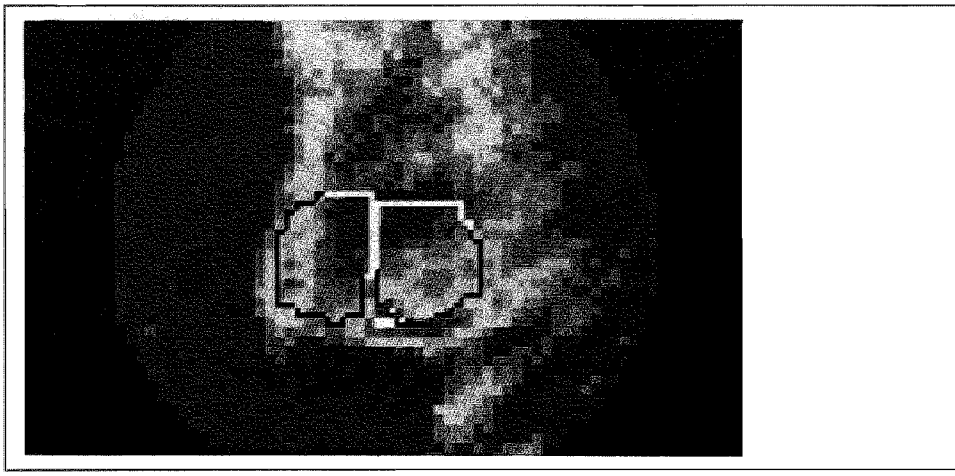

Figure 1 The 45° left anterior oblique view upon which is superimposed the region of interest drawn around the right and left ventricle. No involvement of the right ventricle is seen.

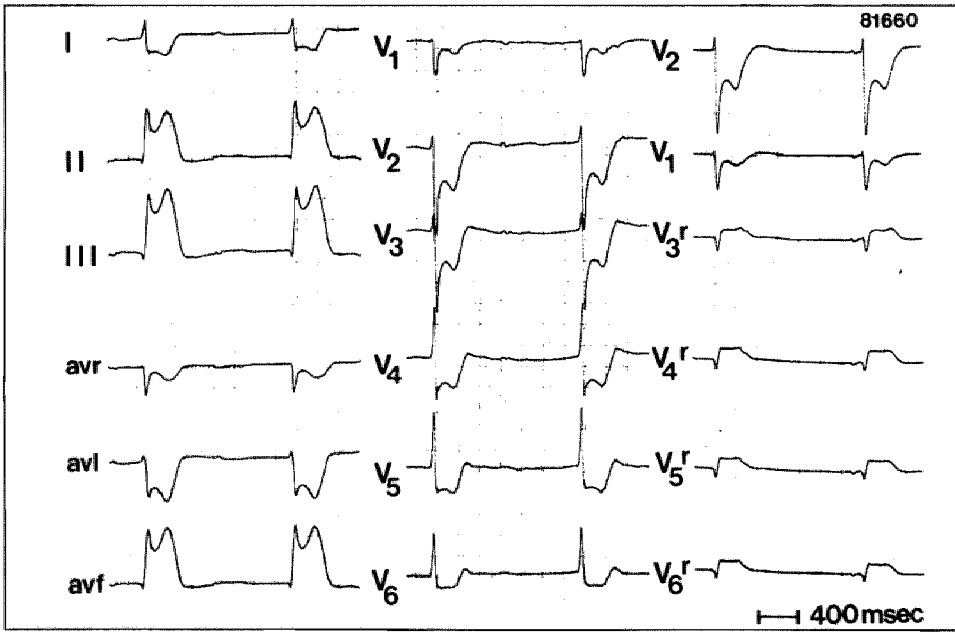

Figure 2 Leads I, II, III, AVR, AVL, AVF, V_1, V_2, V_3, V_4, V_5, V_6 and leads V_2, V_1, V_3R, V_4R, V_5R and V_6R are recorded simultaneously. The electrocardiogram shows an acute infero posterior infarct and a high degree AV nodal block. Note the ST-segment elevation in the right precordial leads indicating right ventricular involvement and the QS-pattern in V_4R and ST-segment depression in V_1 and V_2.

Results

Incidence of second and third degree AV block.

All 67 patients had a positive 99mTechnetium pyrophosphate scan, indicating the presence of an inferior MI. Twenty-nine (43%) of the 67 patients also had involvement of the RV.

Nineteen of the 67 patients had an episode of second or third degree AV block (28%). In 5 patients this was already present on admission. In 9 it developed 3 to 24 hours after admission and in 5 within the next 4 days. Therefore in 14 of our patients the conduction disturbances developed within 24 hours after the onset of chest pain. Block did not disappear after an intravenous injection of 0.5 mg of Atropine. High degree AV nodal conduction disturbances were noted during the first 3 days of continuous monitoring in only 5 of the group of 38 patients (13%) with MI by ECG, but with no RV involvement by pyrophosphate scintigraphy. On the other hand, high degree AV nodal block occurred in 14 out of 29 (48%) of patients with inferior MI by ECG who also had pyrophosphate scintigraphy indicating RV involvement.

In 10 patients a temporary pacemaker was inserted, 8 of them had RV involvement. One patient developed ventricular fibrillation during the introduction of the catheter. This patient had RV involvement on the 99mTechnetium scintigraphy. Only one of the 67 patients died in hospital. This occurred suddenly on the third day after admission.

At autopsy cardiac rupture was found with severe three vessel disease and a recent inferior wall infarction with RV involvement. This patient had a scintigram positive for RV involvement.

Observations in lead V_4R

In 29 patients ST-segment elevation $\geqq$ 1 mm was found in the right precordial lead V_4R (Table 1). In 27 of these patients RV involvement was found on the Technetium pyrophosphate scan. Twelve of the 14 patients with AV conduction disturbances had a scintigram positive for RV infarction and ST-segment elevation in V_4R. Two patients with ST-segment elevation in lead V_4R developed AV conduction disturbances in the absence of RV involvement on the scan.

These data indicate that ST-elevation in lead V_4R not only has the same predictive value for the development of high degree AV conduction disturbances (48%) in patients with an inferior MI, but also is a very sensitive (0.93) and specific (0.95) indicator for RV involvement.

Of interest is the high incidence of AV conduction disturbances in patients with ST-segment elevation in V_4R and ST-segment depression in V_1 and V_2 (7 of 9 patients, table 1) and also that the two patients with an old anterior wall infarction develop conduction disturbances.

Table 1

Morphology in V_4R, V_1 and V_2	In normal AV conduction N = 48	In AV conduction disorders N = 19
ST elevation V_4R	15 (15)	14 (12)
QS in V_4R	10 (8)	11 (8)
ST elevation V_4R and QS in V_4R	8 (8)	9 (8)
ST elevation V_4R and ST depression in V_1 and V_2	2 (2)	7 (7)

Abbreviations: AV = Atrio ventricular nodal. The numbers in parenthesis: Technetium pyrophosphate scan positive for right ventricular involvement.

Discussion

RCA and AV Node.

Inferior wall infarction can result either from an occlusion of the RCA, of the circumflex branch from the left coronary artery, or of the left anterior descending coronary artery if it comes over the apex. Blood supply to the AV node is by way of the AV nodal artery. In 90% of cases this AV nodal artery comes from the RCA. In a varying percentage of cases however, there is a dual blood supply to the AV node, because not only the AV nodal artery but also branches of the first and second septal branches of the left anterior descending artery may bring blood to the AV node (6,7).

AV conduction disturbance with acute inferior MI.

If one accepts that RV involvement in inferior MI will rarely occur following occlusion of the left coronary artery, but primarily after occlusion of the RCA proximal to the RV branch, which is above the origin of the AV nodal artery, one would expect the incidence of AV nodal conduction disturbances to be close to 90% in patients with RV involvement.

Although the incidence of AV block was higher it was however, not as high as expected. An explanation therefore could be that dual or collateral blood supply to the AV node is functionally common.

In our group we found a much higher incidence of AV nodal conduction disturbances in patients with RV involvement, 48% as compared to 13% in patients without RV involvement.
An alternative for the AV nodal conduction disturbances would be the stimulation of the vagal afferents in the inferior wall of the RV and left ventricle. In this case one would expect a satisfactory response to atropine and a higher incidence of sinusbradycardia, which was not the case in our group.
In our series (table 1) we found an interesting subgroup of 9 patients in whom AV nodal conduction disturbances developed in 7. All showed ST-segment elevation in V_4R and ST-segment depression in V_1 and V_2. One explanation for this could be, that the blood supply by the RCA as well as the left anterior descending to the AV node is impaired. This thesis is supported by the fact that both two patients with an old anterior wall infarction developed AV nodal conduction disturbances in contrast to the two patients with an old inferior wall myocardial infarction.

RV infarction with failure.

Interest in RV infarction increased after Cohn et al (8) published their observations on the possible hemodynamic consequences of RV infarction and their therapeutic implications. Recently Lloyd et al (9) reported, that in the presence of an inferior MI with hemodynamic signs of RV involvement high degree AV nodal block occurred in 58% of cases.

VF with cardiac pacing.

Sclarovsky et al (10) described induction of ventricular fibrillation during introduction of a pacing catheter on cardiac pacing in patients with RV infarction. In our patients VF developed in one of the 10 patients in whom a pacing catheter had to be inserted because of symptomatic bradycardia. It was 1 of the 8 patients with RV involvement. In view of the high incidence of RV infarction in patients developing high degree AV nodal block in the setting of an inferior MI our observations suggest that these patients are not at high risk for developing VF during introduction of a cardiac catheter on cardiac pacing.

Abnormal V_4R.

Barrillon et al (11), were the first to recognize the value of right precordial leads in identifying a group with high risk of development of high degree AV block. In their observations the presence of a QS pattern and ST elevation in V_4R did identify a subgroup of patients with high risk of development of severe AV conduction disturbances.

It also has previously been reported that RV involvement can be diagnosed by recording additional RV leads (12-15). In this study we found that ST-segment elevation in lead V_4R has a sensitivity and specificity in predicting RV involvement of 0.93 and 0.95 respectively. We conclude that diagnosing RV infarction by using lead V_4R is of value in discovering a subgroup of patients with inferior MI at high risk of developing high degree block in the AV node even in small RV without hemodynamic RV failure.

We thank Frans J. Wackers, MD, Department of Internal and Nuclear Medicine, Hospital of Vermont, Burlington, Vermont, for support and advice during the preparation of this article.

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Chapter 8

Right and left ventricular ejection fraction in acute inferior wall infarction with or without ST-segment elevation in V_4R

by

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Presented in part at the 31th Annual Meeting of the *Society of Nuclear Medicine in Los Angeles, 1984*

Accepted for publication in the Journal of the American College of Cardiology

Summary

To detect right ventricular involvement lead V_4R was recorded within 10 hours of onset of chest pain in 42 consecutive patients admitted with an inferior wall myocardial infarction (MI). One week after the acute MI multigated equilibrium radionuclide ventriculography was performed to assess right (RV) and left ventricular (LV) ejection fraction (EF). Two weeks after the acute MI coronary angiography was performed to determine the site and location of the obstruction, leading to the acute MI. Seventeen patients had an obstruction in the right coronary artery (RCA) proximal to the first branch to the RV free wall (group I). All had shown ST-segment elevation in lead V_4R. Fourteen patients had an obstruction in the RCA distal to the first branch to the RV free wall (group II). Only 2 of these patients had ST-segment elevation in lead V_4R. In 11 patients the obstruction was located in the circumflex coronary artery (group III). None of these patients had ST-segment elevation in lead V_4R. Nineteen patients had ST-segment elevation $\geqq$ 1 mm in lead V_4R (group IV). LVEF was not different in these groups of patients, while the RVEF was significantly lower in the group of patients with a proximal obstruction in the RCA and in the group of patients previously having ST-segment elevation $\geqq$ 1mm in lead V_4R.
Conclusion: ST-segment elevation in lead V_4R reliably identifies the group of patients with depressed right ventricular function. This persists for at least one week.

Acute inferior wall myocardial infarction (IWMI) can be the result of occlusion of the right coronary artery (RCA), circumflex coronary artery (CA) or a long left anterior descending coronary artery (LAD) extending over the apex of the left ventricle to the inferior wall. Occlusion of such a long LAD may lead to an electrocardiographic pattern of both anterior and IWMI. The differentiation between occlusion of the RCA or CA cannot be made on the standard 12-lead electrocardiogram (ECG). It can, howver, be done when additional right precordial leads and particularly lead V_4R are recorded (1). It has been shown (2-5) that recording the right precordial lead V_4R in patients with acute IWMI allows for recognition of involvement of the right ventricle (RV) in addition to left ventricular (LV) infarction. Two groups of patients can be differentiated by recording lead V_4R during the acute phase of IWMI:

a. Those showing ST-segment elevation in lead $V_4R \geqq 1$ mm. In these patients a proximal occlusion of the RCA before the take off of the first RV branch is extremely likely (1).
b. patients without ST-segment elevation in lead V_4R. In these patients IWMI can be the result of either a distal occlusion of the RCA, or an occlusion of the CA.

Following acute MI the value of left ventricular ejection fraction (LVEF) has important prognostic significance (6, 7). It is conceivable that the degree of impairment of right and left ventricular function in acute myocardial infarction is related to the site and level of the stenosis in the coronary artery (RCA or CA) responsible for infarction.

In this study we therefore prospectively evaluated RV and LV function in patients with IWMI in relation to the vessel responsible for the infarction. In addition the predictive value of ST-segment elevation in right chest lead V_4R to diagnose right ventricular dysfunction was analysed.

Patients and methods

Forty-two consecutive patients were included in this study. Ages ranged from 33 to 69 (mean 54) years. All patients fulfilled the following criteria:

1. typical history of chest pain, lasting more than 30 minutes,
2. admission within 10 hours of chest pain,
3. characteristic cardiac enzyme pattern of serum glutamic oxaloacetic transaminase and creatine phosphokinase for acute myocardial infarction
4. characteristic electrocardiographic changes for acute inferior wall myocardial infarction: serial ST-T-wave changes and the development of Q-waves in lead II, III and AVF.

On admission and every 8 hours during the next 3 days a standard 12-lead ECG and an additional right chest wall lead V_4R were recorded. At the time of the ECG-recordings blood was taken to determine the serum levels of creatinephosphokinase and SGOT.

Approximately 7 days after onset of acute MI, multigated equilibrium cardiac blood pool imaging was performed using in-vivo labeling of autologous red blood cells with 15 mCi of 99mTechnetium (8).

Images were acquired using a Philips gamma camera with a parallel-hole-all purpose collimator, interfaced to a dedicated Philips minicomputer. Data acquisition was performed in that left anterior oblique position in which the best separation between the right and left ventricles was achieved. Left and right ventricular ejection fractions (LVEF) were calculated using a semi-automatic commercial available soft ware program, which has previous been validated by Standke et al (9).

The normal value for LVEF in our laboratory is $\geqq 60\%$ and for the RVEF $\geqq 40\%$.

Ten to 14 days (mean 12.5) after the MI a cardiac catheterisation was performed including a left ventriculography and right and left coronary artery angiography. A reduction of $\geqq 50\%$ in the luminal diameter of a coronary artery was considered significant. The coronary angiograms were judged by two cardiologists. Differences in opinion were resolved by consensus.

The coronary artery demonstrating a complete obstruction or subtotal luminal narrowing was assumed to be the "infarct vessel". In the RCA the location of a stenosis was recorded relative to the take off of the first branch to the right ventricular free wall. No attention was paid to the conus branch.

Results (Table 1)

Angiographic data

In 31 patients the RCA was the "infarct vessel", in 17 the occlusion was proximal (group 1), in 14 distal (group II) to the first branch to the free wall of the right ventricle. In 11 patients the infarct vessel was the CA (group 3).

Electrocardiographic findings on admission and "infarct vessel"

Nineteen out of 42 patients (45%) showed ST-segment elevation ≧ 1 mm in lead V_4R (group 4). All 19 patients had occlusion of the RCA. In 17 patients (89%) the occlusion was located proximal to the first RV branch, while in the 2 other patients the stenosis was located in the RCA distal to the first RV branch, but proximal to a second RV branch. Twenty-three patients had no ST-segment elevation. In 12 patients the stenosis was located in the RCA. In none of these patients the occlusion was located proximal to the first RV branch. None of the 11 patients with an occlusion in the CA, had ST-segment elevation in V_4R. In none of the 42 patients ST-segment elevation was seen in the precordial leads V_1 to V_5.

Table 1

Left and right ventricular ejection fraction. Number of vessels showing significant stenosis and the location of the lesion causing IWMI

No	LVEF	RVEF	No. of Cor.A with stenosis ≧ 50%	Location of the stenosis or occlusion which caused the MI	V_4R
1.	64	27	1	prox. RCA	+
2.	52	48	1	distal RCA	–
3.	66	35	1	prox. RCA	+
4.	45	43	3	CA	–
5.	48	43	3	CA	–
6.	66	44	2	distal RCA	–
7.	59	47	2	distal RCA	–
8.	50	19	3	prox. RCA	+
9.	60	40	2	CA	–
10.	43	44	3	distal RCA	–
11.	49	28	2	prox. RCA	+
12.	51	30	1	prox. RCA	+
13.	57	46	1	distal RCA	–
14.	46	45	1	CA	–

No	LVEF	RVEF	No. of Cor.A with stenosis ≧ 50%	Location of the stenosis or occlusion which caused the MI	V_4R
15.	65	42	1	CA	–
16.	54	42	3	distal RCA	–
17.	54	40	2	CA	–
18.	60	40	2	distal RCA	–
19.	52	44	1	CA	–
20.	55	44	1	distal RCA	–
21.	55	43	3	CA	–
22.	55	33	2	prox. RCA	+
23.	70	42	3	prox. RCA	+
24.	43	47	3	CA	–
25.	50	30	1	prox. RCA	+
26.	55	33	2	prox. RCA	+
27.	59	42	3	distal RCA	–
28.	61	44	3	distal RCA	–
29.	45	31	3	distal RCA	+
30.	58	23	1	prox. RCA	+
31.	47	23	2	prox. RCA	+
32.	62	44	3	CA	–
33.	63	46	3	distal RCA	–
34.	59	16	1	prox. RCA	+
35.	55	14	2	prox. RCA	+
36.	45	37	2	distal RCA	+
37.	63	50	2	CA	–
38.	53	26	2	prox. RCA	+
39.	61	48	1	prox. RCA	+
40.	62	48	3	distal RCA	–
41.	44	31	3	prox. RCA	+
42.	50	27	1	prox. RCA	+

$\bar{x}$ LVEF = 53.3 ± 10
$\bar{x}$ RVEF = 37.4 ± 10

Abbreviations:
LVEF = left ventricular ejection fraction; RVEF = right venricular ejection fraction; prox = proximal to the first branch to the right ventricle; RCA = right coronary artery; CA = circumflex coronary artery; IWMI = inferior wall myocardial infarction. Cor.A = Coronary Arteries.

LVEF and RVEF and "infarct vessel" (Table 2)

LVEF ranged from 36 to 70% (mean 53% ±10) and RVEF ranged from 14 to 50% (mean 37% ±10) for the whole group of patients. In 17 patients with a proximal RCA occlusion (group 1), mean LVEF was 55 ±7% (mean + s.d.). Mean RVEF value was 29 ±9%.

In 14 patients with a distal RCA occlusion mean LVEF (group 2) was 56 ± 7% and mean RVEF was 43 ± 5%.
In 11 patients with an occlusion in the CA (group 3) mean LVEF was 53 ± 9% and RVEF was 44 ± 3%.
There was no statistically significant difference in mean LVEF between the 3 groups. Mean RVEF was significantly lower in patients of group 1, compared to group 2 and 3 ($p \leq 0.001$) according to the unpaired Student T-test.

LVEF and RVEF and electrocardiographic findings

Of 19 patients with ST-segment elevation in V_4R during the acute phase of myocardial infarction 17 had abnormal RVEF seven days later. Mean RVEF in this group was 29% ±8. All 23 patients without ST-segment elevation in V_4R had normal RVEF. Mean RVEF in this group was 44% ±3 ($p \leq 0.001$). Of 19 patients with ST-segment elevation in V_4R 14 patients had a depressed LVEF. Mean LVEF in this group was 54% ±7. Fourteen out of 23 patients without ST-segment elevation in lead V_4R had an abnormal LVEF (p = NS). The mean LVEF in this group was 56% ±7.

One patient (no. 31) died. On admission this patient had a total atrio-ventricular block and he only showed signs of right sided heart failure. The central venous pressure was elevated. The blood pressure was 70/40 mmHg. With a Swan-Ganz catheter the pressures in the right side were measured. The end diastolic pressure in the right ventricle was twice as high as the wedge pressure. After a fluid load and insertion of a temporary pacemaker the hemodynamic situation improved rapidly. Three weeks after the acute MI the patient died suddenly. Post mortem examination revealed severe three vessel disease and signs of an IWMI with an enormously dilated right ventricle (Fig. 1). However, there were no signs of a reinfarction, suggesting that he suffered from a fatal arrhythmia.

Figure 1 The heart of the only patient who died three weeks after the acute MI. As you can see the RV is markedly dilated while the left ventricle looks normal.

Table 2

LVEF and RVEF in the different groups of patients studied

	LVEF	RVEF
Occlusion proximal to the first branch to the *right ventricle* in the RCA (N = 17)	55 ±7%	29 ±9%
Occlusion distal to the first branch to the right ventricle in the RCA (N = 14)	56 ±7%	43 ±5%
Occlusion in the circumflex coronary artery (N = 11)	53 ±9%	44 ±3%
ST-segment elevation ≧ 1mm in V_4R (N = 19)	54 ±7%	29 ±8%
No ST-segment elevation in lead V_4R (N = 23)	56 ±7%	44 ±3%

Abbreviations: See table 1.

Discussion

The present study demonstrates that ST-segment elevation in V_4R observed during the acute phase of inferior wall myocardial infarction is indicative of depressed global right ventricular function. The findings also confirm our previous observation that as a rule patients with ST-segment elevation in V_4R have a proximal stenosis of the RCA. Therefore, a simple and inexpensive tool, such as the electrocardiogram, provides information that may be critical for appropriate clinical management of patients with inferior wall MI. Our data do not support the data of Geft et al (8) while in none of the 42 patients ST-segment elevation in the precordial leads was observed. In the present study only 1 of 17 patients with a depressed RVEF has clinical signs of right sided heart failure. Thus the right ventricular dysfunction often is clinically not noticed and appears to have no short term prognostic significance. In the whole study population the LVEF was well preserved (mean LVEF 53% ± 10). In the 17 patients with a depressed RVEF, 14 also had depressed LVEF, while in the 25 patients with a normal RVEF 14 patiens had a depressed LVEF. The calculation of the right ventricular ejection fraction in a multigated study is complicated by the changing overlap of the right atrium. Because there are no reasons to believe that there is a difference between the overlap of the right atrium in these groups we believe that we are justified in comparing the value of RVEF derived by multigated nuclear techniques in these groups.

Whether or not changes in RV-function after IWMI have indeed independent long term prognostic significance, can only be answered by a long follow up study of these patients. Also in regard to follow up other questions remain presently unanswered, such as in what percentage of RVI develops right sided failure? How extensive must RVI be in these patients?

Our study does not make clear what amount of damage to the RV is required to result in the clinical picture of RVI. Although the incidence of this picture is small, early recognition and appropriate treatment is important because treatment of cardiogenic shock in this setting is very different from patients having shock from massive involvement of the LV (10).

When RV involvement during IWMI does not result in hemodynamic problems one is inclined to believe that a minor part of the posterior septum and inferior wall of the RV led to the ECG-changes in the right precordial leads. Because of the uncomplicated course of these patients one might also believe that no or very minimal abnormalities in RV function are present. Our study shows that occlusion of the RCA in patients before the RV branch severe depression of the RVEF is present. While none of these patients (except for patient 31) had clinical signs of right-sided heart failure. However, in our department hemodynamic monitoring is only performed when a patient is in a bad hemodynamic condition. So hemodynamic abnormalities could have been present in those patients with right ventricular infarction. Our find-

ings indicate that in all patients with ST elevation in V_4R permanent damage to the RV had been produced during acute MI.

Acknowledgement

We express our appreciation to Serve Halders, Irene Cajot and Gerda Saat for their technical and to Miep Schrooders for her secretarial assistance.

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Chapter 9

Conclusions

This thesis reports on the value of lead V_4R to obtain information about right ventricular involvement and possible complications in the setting of severe ischemia or infarction of the inferior wall of the myocardium. The following conclusions can be drawn:

1. Using technetium 99m pyrophosphate imaging to diagnose right ventricular involvement, in patients with acute inferior myocardial infarction the sensitivity, specificity and predictive accuracy of lead V_4R to detect right ventricular involvement was found to be 93%, 95% and 93% respectively.
2. All patients with a subtotal or total stenosis in the right coronary artery proximal to the first branch to the right ventricle showed ST-segment elevation $\geqq$ 1 mm in lead V_4R. No ST-segment elevation in lead $V_4R \geqq 1$ mm was seen, however, in patients with an acute inferior wall myocardial infarction caused by an occlusion of the left circumflex coronary artery.
3. As shown in a case report the observations described under 2 can have important implications when emergency procedures are necessary in the patient with an acute inferior wall myocardial infarction.
4. ST-segment deviation $\geqq$ 1 mm in lead V_4R during exercise testing strongly suggests a critical stenosis in the proximal part of the right coronary artery. In patients without an inferior wall infarction this finding has a sensitivity of 50%.
5. ST-segment elevation in $V_4R \geqq 1$ mm in the setting of an acute inferior wall myocardial infarction is of great importance to recognize patients at risk for developing high degree AV-block. Approximately 50% of patients with an acute inferior wall myocardial infarction and ST-segment elevation $\geqq$ 1 mm developed atrio-ventricular-nodal conduction disturbances, while this was only seen in 13% of patients with an acute inferior wall myocardial infarction without ST-segment elevation $\geqq$ 1 mm in lead V_4R.
6. Patients with inferior wall infarction showing ST-segment elevation in V_4R during the acute phase have significantly lower right ventricular ejection fractions than patients without this finding. In summary, the finding of ST-segment elevation $\geqq$ 1 mm in lead V_4R in the setting of an acute inferior wall myocardial infarction is of value, because it provides information on the site of obstruction, prevalence of subsequent AV nodal block, and right ventricular dysfunction. Therefore, lead V_4R should be recorded in every patient with an acute inferior wall myocardial infarction. The registration of lead V_4R during exercise testing is useful to recognize a critical stenosis in the right coronary artery.

Summary

Following the introduction, the methods section describes the experimental techniques used in this study. The placement of the right chest wall leads, the normal electrocardiographic pattern in these leads and, the measurement of ST-segment deviation are discussed. Two radionuclide techniques are described:

1. *Pyrophosphate imaging.* The principles, procedures and limitations of this technique for the detection of right ventricular infarction are discussed together with the use of bolus radionuclide injection to localize the right ventricular wall.
2. *Multigated cardiac blood pool imaging.* The imaging techniques, data processing methods and means of calculating right and left ventricular ejection fractions are described. The validation and limitation of this technique are also discussed.

The methodology section also describes the determination of LVEF and coronary artery stenosis by contrast angiography. In chapter 3 the value of the electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction is described. We found that ST-segment elevation $\geqq$ 1 mm in lead V_4R was a sensitive and specific sign for right ventricular involvement when compared with technetium pyrophosphate imaging as the "gold standard".
Chapter 4 describes the value of lead V_4R recording for recognition of the occluded vessel in patients with acute inferior wall myocardial infarction.
The absence of ST-segment elevation excludes proximal occlusion of the right coronary artery while ST-segment elevation $\geqq$ 1 mm in lead V_4R has a 100% sensitivity to predict proximal occlusion in this vessel. Chapter 5 is a case report emphasizing the clinical value of detecting ST-segment elevation $\geqq$ 1 mm in lead V_4R in a patient with previous bypass surgery. The recognition of right coronary graft occlusion enabled immediate and successful streptokinase therapy.
In chapter 6 the value of recording lead V_4R during exercise testing in predicting proximal stenosis in the right coronary artery is described. The specificity of ST-segment elevation $\geqq$ 1 mm in lead V_4R in predicting or excluding proximal stenosis in the right coronary artery is very high at 95%. The sensitivity is 56% giving an overall predictive value of 84%.
Chapter 7 describes the clinical significance of ST-segment elevation $\geqq$ 1 mm in V_4R during the acute stage of inferior wall myocardial infarction. The prevalence of subsequent atrioventricular nodal conduction disturbances is significantly higher in patients with this indicator of proximal right coronary occlusion.
Chapter 8 describes a study of the right and left ventricular ejection fraction in patients with acute inferior myocardial infarction. In those with ST-segment elevation $\geqq$ 1 mm in V_4R the RVEF was significantly lower than in the group without the electrocardiographic abnormality. There were no differences in mean LVEF between the two groups.

Samenvatting

Na een introductie worden de methoden, die gebruikt worden, beschreven. Allereerst wordt aangegeven hoe de plaatsing van de rechts precordiale afleidingen is. Hoe het normale patroon van deze afleidingen is en de manier waarop ST-segment elevaties worden gemeten. Vervolgens worden de radionucleaire technieken beschreven. Eerst hoe met behulp van Technetium pyrofosfaat de localisatie van het hartinfarct plaats vindt en vervolgens hoe met behulp van een bolustechniek vastgesteld wordt of de rechter ventrikel ook in het infarctgebied is betrokken. Ook wordt aandacht geschonken aan de beperkingen van deze techniek. Daarna wordt de multigated cardiac blood pool imaging beschreven, met name de opnametechniek, de bewerkingsmethode en de wijze waarop de berekening van de linker en rechter ventrikel ejectiefractie plaats vindt. Ook bij deze techniek worden de beperkingen beschreven. Voorts vindt er een validatie van deze techniek plaats. Het laatste deel van de methodologie betreft de angiografische technieken; hoe de linker ventrikel ejectiefractie berekend wordt met behulp van een angiogram, en de wijze waarop coronair angiogrammen beoordeeld worden.

In hoofdstuk 3 wordt de waarde van het electrocardiogram om rechter ventrikelinfarcering vast te stellen bij patienten met een acuut onderwandinfarct beschreven. Vastgesteld werd dat ST-segment $\geqq$ 1 mm in afleiding V_4R een zeer gevoelig en specifiek teken is voor rechter kamerinfarcering.

Hoofdstuk 4 beschrijft de waarde van afleiding V_4R voor het herkennen van die kransslagader, die verantwoordelijk is geweest voor het acute onderwandinfarct. De afwezigheid van ST-segment elevatie in afleiding V_4R sluit een proximale afsluiting van de rechter coronair arterie uit, terwijl ST-segment elevatie $\geqq$ 1 mm in afleiding V_4R een gevoeligheid van 100% heeft om een proximale stenose in de rechter coronair arterie te voorspellen.

Hoofdstuk 5 is een ziektegeschiedenis, waarbij de waarde van ST-segment elevatie $\geqq$ 1 mm in afleiding V_4R bij een patient na bypasschirurgie wordt beschreven. Nu waren we in staat onmiddellijk de afgesloten bypass te injecteren met streptokinase.

In hoofdstuk 6 wordt de waarde van afleiding V_4R bij inspanning beschreven om een proximale afsluiting in de rechter coronair arterie vast te stellen. De specificiteit van ST-segment deviatie $\geqq$ in afleiding V_4R is zeer hoog (96%), terwijl de gevoeligheid 56% bedroeg.

Hoofdstuk 7 beschrijft de klinische waarde van ST-segment elevatie $\geqq$ 1 mm in afleiding V_4R tijdens het acute stadium van een onderwandinfarct. Bij patienten met ST-segment elevatie $\geqq$ 1 mm in afleiding V_4R treden atrioventriculaire geleidingsstoornissen beduidend vaker op dan bij die groep van patienten waarbij deze ST-segment elevatie in V_4R niet wordt gezien.

In hoofdstuk 8 worden de verschillen in rechter en linker ventrikel ejectiefractie bij patienten met een acuut onderwandinfarct met of zonder ST-segment elevatie $\geqq$ 1

mm in V_4R beschreven. Het wel of niet aanwezig zijn van ST-segment elevatie in V_4R beïnvloedt de linker ventrikel ejectiefractie niet. Wel werd een significante lagere ejectiefractie van de rechter ventrikel gevonden bij die patienten, waarbij een ST-segment elevatie in V_4R was gevonden tijdens het acute stadium van het hartinfarct.

Curriculum vitae

Simon Hubertus Joseph Gerardus Braat was born on March 17, 1948 in Roosendaal, The Netherlands. He graduated from high school St. Norbertus Lyceum, Roosendaal in 1966. In 1967 he went to Medical School at the Free University of Amsterdam and graduated in March 1974. From May 1974 till July 1975 he served Her Majesty Queen Juliana of The Netherlands in military service. In August 1975 he started his residency in Internal Medicine at the Department of Internal Medicine of het Grootziekengasthuis in 's Hertogenbosch (Head Dr.J.B. Lips). In November 1977 he began his cardiologic training at the Department of Cardiology at St. Annadal Hospital in Maastricht under supervision of Prof. Dr. H.J.J. Wellens. From January 1980 till July 1980 he worked as a research fellow in Nuclear Cardiology at the Department of Cardiology at the Yale University, New Haven, Connecticut, U.S.A. (Head: Prof. Barry Zaret).
In November 1980 he completed his cardiologic training and became a member of the Capaciteitsgroep of Cardiology of the University of Limburg with special interest in nuclear cardiology.

Acknowledgements

Many people have supported me in the work which lead to this thesis and the associated publications. It is impossible to thank everyone in person, but I want to express special thanks to those without whom I could not have started this research.
Prof. Hein Wellens, your scientific and dynamic personality created the infrastructure of a Department of Cardiology that gave me the opportunity to spend 6 months at Yale University in Connecticut to learn the basic principles of nuclear cardiology and thereafter to use this knowledge to start scientific investigations. I am deeply grateful for your encouragement and guidance.
Prof. Frans Wackers, I shall always remember the hospitality of you and your wife Marianne. You provided me with every opportunity to make my stay in New Haven a fruitful one. Without the help of your family, not only I, but also my wife Anneke, my children Jeroen en Manon, could not have enjoyed the six months in New Haven as much as we did. After my return to Maastricht you were always available to give advice and your remarks undoubtedly added to the quality of this thesis.
Prof. Meyler and Prof. Flendrig, I greatly appreciate your critical reading and advice.
Dr. Pedro Brugada, the encouragement you gave me to continue my work and the stimulating discussions were of great value to me.
Miep Schrooders, I thank you very much for your patience and skill in typing and changing the manuscript and taking care that I remained working on this thesis.
Last but not least, I want to mention Anneke, Jeroen and Manon for their endurance and understanding allowing me to continue my study. Maybe we can soon make up for this.